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AMOEBIASIS AND DYSENTERY.

AMOEBIASIS.

PHILIPTSCHENKO (A. A.). **The Occurrence of Intestinal Protozoal Infections in the Inhabitants of Leningrad (U.S.S.R.) with Special Reference to** *Entamoeba histolytica* **Carriers.**—*Ann. Trop. Med. & Parasit.* 1930. July 8. Vol. 24. No. 2. pp. 165–175.

Six hundred and four persons were examined in Leningrad for intestinal parasites, one examination in each case. Amongst the group of 400 food-employees, 262 (65·5 per cent.) were found infected with various protozoa; *E. histolytica* was discovered in 91 cases (22·75 per cent.); 189 (47·25 per cent.) of these employees suffered from various gastro-intestinal disorders. Of 162 adult non-dysenteric patients treated in the dispensary for various gastro-intestinal disorders, 101 (62·4 per cent.) were found to be infected with various protozoa; *E. histolytica* was discovered in 41 cases (25·3 per cent.). An investigation of 42 child dispensary patients suffering from various gastro-intestinal disorders showed that 20 were infected with various protozoa. In four cases *E. histolytica* infections were found.

The evidence resulting from the measurement of *E. histolytica* cysts rather contradicted BRUMPT'S suggestion of the existence of non-pathogenic entamoebae with small four-nucleated cysts. On the other hand, the presence in half of the cyst-cases of different sizes of cysts seemed to confirm the view of DOBELL, BOECK, and others as to the existence of different races of one *E. histolytica.*

H. M. Hanschell.

PHILIPTSCHENKO (A. A.). *Entamoeba histolytica* **and Other Protozoa in Two Hundred and Twenty-five Cases of Acute Aestival Diarrhoea admitted to the Botkin Barracks Hospital at Leningrad (U.S.S.R.).**—*Ann. Trop. Med. & Parasit.* 1930. July 8. Vol. 24. No. 2. pp. 177–187. [3 refs.]

Only those patients were noted as harbouring *Entamoeba histolytica* who disclosed: (1) Entamoeba containing red blood corpuscles, (2) cysts of *E. histolytica* (associated or not associated with motile stages), (3) motile stages of Entamoeba not containing red blood corpuscles, no cysts present, but the Entamoeba characterized by complete absence of ingested material, by sharp differentiation between endo- and ecto-

plasm, or by a nucleus showing the typical features of a delicate nuclear membrane with single layer of minute chromatin grains on its inner surface, a minute karyosoma situated in centre of nucleus, and no chromatin between karyosoma and nuclear membrane. All doubtful cases not showing clearly the above characters were noted as *E. coli.*

From July 1st to August 31st, 225 patients with various acute intestinal affections were examined, and 110 (49·0 per cent.) were found infected with various protozoa. *E. histolytica* was found in 32 cases (14·2 per cent.) ; in 13 of these (5·8 per cent.) the *E. histolytica* contained red blood corpuscles. As 173 cases were examined only once each, the real figures of *E. histolytica* infection should be considerably increased ; it may be said that about 15 to 20 per cent. of acute aestival diarrhoea cases are true amoebic dysentery. Bacillary dysentery amongst these 225 cases was found in 22 (9·8 per cent.) and only in one case was it combined with amoebic. Prevalence of intestinal protozoa in general, and of *E. histolytica* especially, makes a protozoological examination of all acute intestinal disorders absolutely indispensable.

H. M. H.

CUSUMANO (A.). La diffusione dell' "Entamoeba histolytica" in Sicilia. [**The Spread of** *Entamoeba histolytica* **in Sicily.**]—*Policlinico.* Sez. Prat. 1930. Oct. 13. Vol. 37. No. 41. pp. 1489–1490.

A recent promulgation of the health authorities of Palermo calling the attention of medical men to the existence of amoebiasis in that Province has led the author to state that the condition is of equal severity in Trapani, Caltanissetta, Agrigento, and in fact over most of the island. The symptoms produced are : in children dysentery, in adults spastic colitis and diarrhoea alternating with constipation. Less common are the appendicular syndrome, symptoms resembling those of cholecystitis, or duodenal trouble. [The value of this paper is somewhat discounted by the statement that " vegetative forms of the amoeba have been met with in various organs besides the liver, such as the brain, lung, spleen, pericardium, heart, joints, urinary system, lymph glands and peripheral vessels," adducing no proof of this extraordinary dictum.]

H. H. S.

CRAIG (Charles F.). **The Diagnostic Value of the Complement Fixation Test in Amebic Infections.**—*Jl. Amer. Med. Assoc.* 1930. July 5. Vol. 95. No. 1. pp. 10–13. [8 refs.] [Army Med. School, Washington.]

In July 1927 the author first called attention to the fact that alcoholic extracts of cultures of *E. histolytica* possessed marked complement binding power when used as antigen in complement fixation tests of individuals infected with that parasite [see this *Bulletin*, Vol. 25, pp. 228 and 623]. In this paper he gives his results obtained with this test up to March 30th, 1930 ; and discusses its value in diagnosis of *E. histolytica* infections based on his experience of it during nearly three years.

Of 689 cases in which the faeces were examined for entamoebae 84 (12 per cent.) gave a positive C.F.T. reaction ; 71 gave a clear-cut 4 plus reaction, and the rest a clear-cut 3 plus reaction. Of these 84

the faeces disclosed *E. histolytica* in 77. In 13 [? 7] the amoeba was not found in one or two examinations.

Six hundred and five cases gave negative C.F.T. reaction. In these, *E. histolytica* was found in the faeces of 5 (0·8 per cent.). One of these five was a severe acute case of amoebic dysentery ; one was an abscess of liver ; one patient was symptomless ; whilst the other two had suspicious gastro-intestinal symptoms.

One hundred and fifty-nine (26·2 per cent.) of the patients giving negative C.F.T. reaction were found to be infested with some species of amoeba other than *E. histolytica.* Thirteen of the 84 patients giving positive C.F.T. reaction with *E. histolytica* antigen also gave positive Wassermann and Kahn reaction ; the remaining 71 gave negative Wassermann and Kahn reactions.

Of the 605 cases giving negative C.F.T. reaction (*E. histolytica*) 54 (9 per cent.) gave positive Wassermann and Kahn reactions ; the remaining 551 (91 per cent.) gave negative Wassermann and Kahn reactions. Thus there were slightly more than 6 per cent. positive Wasserman and Kahn reactions in those giving a positive C.F.T. result with *E. histolytica* antigen than in those giving a negative result. It is also significant that of the 13 individuals giving positive reaction to all 3 tests, no less than eight failed to show *E. histolytica* in the faeces (2 examinations only of faeces). Apparently then in rare instances syphilitics will give a positive reaction with *E. histolytica* antigen. At the present time the practical value of the test is limited by the difficulty in preparing the antigen extract. The technique of the test is rendered more difficult by reason of fact that the antigenic extract is so weak in antigenic properties that it must be used undiluted. Therefore every step in the technique must be thoroughly controlled. The test is therefore still only for laboratories where services of competent serologists and protozoologists are available.

H. M. H.

PENSO (Giuseppe). Sopra un'ameba parassita della vescica urinaria *Entamoeba vesicalis* n. sp. (mihi). **[An Amoeba Parasitic in the Urinary Bladder.**]—*Ann. Med. Nav. e Colon.* 1929. Year 36. Vol. 1. No. 1/2. 24 pp. With 9 figs. on 1 plate. [48 refs.] English summary. [Inst. of Med. Parasit., Univ., Rome.]

After giving a useful review of the previous records of the occurrence of amoebae in urine, the author describes his own case—that of a child, eight years of age. The urine contained numerous deformed leucocytes and swollen and degenerate bladder cells. It gave a heavy deposit of crystals of calcium oxalate. In addition to the cells there were numerous slowly moving amoebae of 20 to 40 microns in diameter. The urine was examined once a week for two months and the amoebae were constantly present in large numbers. Amoebae were absent from the mouth and intestine. In the urine the amoebae remained alive for five to seven days, and multiplication by division could be followed. Uninucleate cysts are also described. The amoebae in the free and encysted stage, as they appear after staining with methylene blue and eosin without fixation, are shown in a plate. [There is nothing to indicate that the encysted stage is what it is supposed to be, and this combined with the fact that the amoebae were unstainable by any of the ordinary methods which readily succeed with every known amoeba leads the reviewer to suspect that degenerate cells may again have been

mistaken for parasites.] On the strength of his observations the author introduces a new name—*Entamoeba vesicalis.* C. M. Wenyon.

VENTURI (Luigi Carlo). Contributo allo studio della cistite amebica. [**Amoebic Cystitis.**]—*Arch. Ital. Sci. Med. Colon.* 1930. Aug. 1. Vol. 11. No. 8. pp. 465–473. With 1 text fig. [10 refs.] English summary (6 lines).

The author gives brief reference to many reports, since that of BAELZ 1883, of urinary amoebiasis. His own case, here reported and discussed in detail, was that of a man 38 years old. Illness began with fever, hypogastric pain, vomiting, followed by frequent micturition; perineal pain; escape of blood from external urinary meatus. Pain and tenderness over bladder. The patient denied any previous venereal disease, or exposure to venereal infection. Urine withdrawn by catheter was very turbid, reddish, slightly acid; sp. grav. 1018; and contained trace of albumen. Microscopical examination of the sediment revealed very few bacteria; no casts; many white cells, some breaking down; very many intact erythrocytes, and also large cells, some protruding large pseudopodia, whose protoplasm contained an indistinct marginal nucleus, and numerous inclusions, especially red blood cells in various stages of digestion; no included bacteria. Some of these cells showed well differentiated ecto- and endoplasm and contained nuclei in various numbers. Careful search failed to detect gonococci or Koch's bacillus. Two guineapigs were intraperitoneally inoculated with the urinary sediment. They were killed after one and two months respectively—no abnormalities were found in their internal organs. The patient's faeces revealed cysts of *Entamoeba histolytica*, no other protozoa and no helminth ova were detected. (The patient had suffered, from time to time, from slight transient attacks of mucosanguineous diarrhoea.) The urinary sediment was injected into the rectum of a kitten; and the kitten's rectum sealed for two days. No amoebic infection of the kitten resulted. [Nevertheless, the author declares] the diagnosis of amoebic cystitis was not in doubt, for besides finding in the fresh material the large vegetative forms and smaller "precystic" "more resistant" forms, examination of smears of the urinary sediment, Schaudinn-fixed, and Giemsa- or iron-haematoxylin-stained, disclosed, in smaller numbers than in the fresh preparations, amoebae possessing the typical structure of *E. histolytica*, with excentric nucleus and chromatin masses disposed discretely on the nuclear membrane. The patient's symptoms had vanished completely after 12 days' treatment by stovarsol, two tablets of 0·25 gm. each per os daily; and vesical lavage with 0·1 per cent. silver nitrate solution.

[The author illustrates this paper with a drawing of the bodies seen in the fresh unstained urinary sediment. The cells, even pseudopodic ones, show no differentiation between endo- and ecto-plasm; and for the rest only characters common to entamoebae and tissue cells. Illustration of the diagnosed amoebae in the wet-fixed and iron-haematoxylin-stained smears would have been more to the point.] H. M. H.

GAMBIER (A.). Un cas de cystite aiguë amibienne. [**Case of Acute Amoebic Cystitis.**]—*Bull. Soc. Path. Exot.* 1930. May 14. Vol. 23. No. 5. pp. 457–458.

The patient, a native of Tonking, developed acute dysentery, diagnosed as amoebic, and rapidly relieved by emetine therapy. A few days later he passed purulent blood-stained urine. The author found in this urine, and on 19 occasions subsequently, large "amoebae," containing red blood cells, and with well-defined ecto- and endo-plasm. These "amoebae" were immobile in centrifuged material, but in uncentrifuged urinary deposit definitely mobile.

The author declares that in the locality, Phnôm-Penh, amoebiasis and trichomoniasis are very common. Thus in one year he has carried out 463 positive examinations for dysentery amoebae, and 209 for trichomonas. He was thus well acquainted with these parasites. He was aware of DESCHIENS' insistence that the amoebic nature of this cystitis should be demonstrated by examination of wet fixed and stained preparations, and by inoculation into kittens ; he had done neither, yet was positive of the amoebic nature of the haematophagous cells—additional evidence in support being that the cystitis and the haematophagous cells disappeared after 3 days' emetine treatment. DESCHIENS discusses the case, and notes that verifiable evidence, such as would be provided by wet fixed stained permanent preparations, as to amoebic nature of the haematophagous cells, is lacking, but suggests possibility of amoebic abscess forming during the dysentery attack in anterior wall of rectum and breaking through the thin prostato-peritoneal aponeurosis into bladder.

H. M. H.

PETZETAKIS. Amibhémie et abcès amibien primitif du poumon sans dysenterie. "La forme amibhémique de l'abcès du poumon." Sur un cas d'abcès primitif du poumon, ayant été précédé d'un long état pyrétique d'allure septicémique pendant plusieurs mois, opéré sans amélioration et guéri rapidement après traitement émétinique. [**Amoebihaemia and Primary Amoebic Abscess of Lung.**]—*Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1930. July 21. Year 46. 3rd Ser. No. 25. pp. 1341–1349. With 6 text figs. [3 refs.]

A man 30 years old suffered for 2 months from fever with morning remission. There was no history of previous intestinal troubles nor did any develop during fever. Repeated examinations of the stools revealed no amoebae. Though on general grounds the case was counted as typhoid fever, repeated examinations of the blood proved negative. Clinical examinations during this prolonged pyrexia failed to detect any abnormality in lungs or in other viscera. Examination of sputa was negative. At end of the two months' fever the patient developed severe pain in right chest and fever became higher. Radiography disclosed a large abscess in right lower lobe of right lung. The patient was sent to hospital, exploratory puncture obtained pus, and operation then evacuated a large quantity. Examination of this pus revealed no amoebae. There was no fall of temperature and no improvement in patient's condition, when 5 days after operation scrapings of the abscess wall revealed amoebae. Emetine injections were given and within 24 hours temperature was normal and patient well on the road to recovery. Healing was complete under emetine treatment, final radiography disclosing apparently normal lung.

The author insists that the case demonstrates :—

(1) preliminary lung amoebihaemia, manifested by fever.

(2) "amoebic localization" by amoebic embolus in lung and rapid abscess formation.

(3) insufficiency of operative treatment by itself.

(4) the rapid influence of emetine.

He records, moreover, that, after operation and emetine therapy, the sputa [apparently found to contain amoebae only after these had been found in the abscess pus] continued to disclose amoebae until after the third course of emetine injections.

H. M. H.

LABBÉ (Marcel). Abcès du poumon probablement amibiasique guéri par l'émétine. [**Abscess of Lung believed Amoebic cured by Emetine.**]—*Presse Méd.* 1930. July 23. Vol. 38. No. 59. pp. 993–994. With 4 text figs. [13 refs.]

The patient had suffered from acute and chronic dysentery acquired in Algiers. There was fever, copious mucopurulent expectoration and leuco-

cytosis of 17,000, and radiography revealed abscess in left upper lobe of lung. No amoebae could be found in sputa or in stools. After other remedies had failed, emetine injections were followed by disappearance of fever and purulent expectoration, and radiography showed resolution of lesion in left lung. The author notes that amoebae were not demonstrated; that many authors have reported cure of pulmonary abscess by emetine where no amoebae could be demonstrated nor an amoebic origin reasonably suspected; nevertheless he endorses the dictum that the touchstone of amoebiasis is emetine therapy.

H. M. H.

HOUSSIAU. Troubles des faisceaux cerebelleux et du cervelet. Conséquence d'une ancienne dysenterie amibienne. Un cas de dysenterie datant de près de 10 ans. [**Cerebellar Symptoms attributed to Old Amoebic Dysentery.**]—*Ann. Soc. Belge de Méd. Trop.* 1930. June 30. Vol. 10. No. 2. pp. 177–180.

1. A woman after 9 years in the Congo developed marked signs of cerebellar lesions—severe vertigo, persistent headaches, continual vomiting, frequent falls always to the left. Eventually *E. histolytica* cysts were found in her faeces. Emetine injections were followed by very definite cessation of cerebellar symptoms. The author declares not for amoebic cerebellar abscess but for amoebic invasion of the brain by *E. histolytica*—this being "démontrée par l'efficacité du traitement par l'emétine"

2. A woman who had never been in tropics had suffered for over nine years from almost continuous muco-sanguineous diarrhoea. Active amoebae were found in her stools. She was cured of symptoms by emetine injections.

H. M. H.

KILBOURNE (E. D.). **Amebic Abscess of the Liver—its Treatment.**—*California & Western Med.* 1930. Sept. Vol. 33. No. 3. pp. 658–661. [9 refs.]

The author states that although amoebic abscess of the liver is met much less frequently since the advent of emetine, it is not uncommon in the United States. Medical treatment by ipecacuanha, emetine, and other drugs prevents many abscesses, but it is insufficient after they have formed. Blind exploration and aspiration by needle are inefficient and dangerous. Laparotomy is necessary, whether the abscess be single or multiple, except when liver is adherent to abdominal wall thereby making needle puncture relatively safe; even then one must guess at what is happening in remainder of liver. Location of abscess or abscesses preliminary to evacuation should be made by intra-abdominal palpation by the whole hand. Many an abscess will thus be found that would be missed by simple puncture. An occasional patient saved is the reward of the extra operative work. The abscess cavity may be drained best by abdominal, subpleural, or transpleural routes after such accurate localization.

H. M. H.

ARAFA (M. A.). **Treatment of Dysentery.**—*Jl. Egyptian Med. Assoc.* 1930. May. Vol. 13. No. 5. pp. 168–197. [9 refs.]

This paper deals with treatment of amoebic dysentery by rivanol. The diagnosis of each case was based on results of careful microscopic examination of material from the gut—either in stool or obtained by

sigmoidoscope, and on sigmoidoscopy—which last was used also to watch results of treatment. The author concludes that yatren enemata combined with yatren pills orally produce rapid healing of amoebic ulcers, and render stools free from *E. histolytica.* The author has tried rivanol and yatren treatment combined in too few cases as yet to warrant expression of opinion as to its value. Rivanol given rectally is irritating to mucosa in 1 : 2,000 concentration, causing marked hyperaemia of rectal mucosa as viewed through sigmoidoscope. In 1 : 10,000 it has no lethal effect on *E. histolytica.* Rivanol given orally in doses as high as 0·075 gm. t.d. has not been found to produce any toxic effects on the patient, nor to have any marked lethal effect on *E. histolytica.* In only 3 very mild amoebic infections did *E. histolytica* disappear from stools ; all other cases soon showed return of symptoms and *E. histolytica* in active vegetative form in stools either before or soon after completion of rivanol course. Rivanol did, however, procure marked relief from tenesmus, and blood and mucus soon disappeared—to return soon after stopping the drug. However, the antispasmodic and antiseptic action of rivanol may with advantage be utilized for relief of colic and tenesmus, and for assisting cleansing of bowel of superadded infection in various dysenteric conditions where lower bowel symptoms are a marked feature of the disease.

H. M. H.

CHOPRA (R. N.) & DE (N.). **A Failure of the Alkaloids of** *Holarrhena antidysenterica* **(Kurchi) in the Treatment of Amoebic Hepatitis.**—*Indian Med. Gaz.* 1930. July. Vol. 65. No. 7. pp. 391–392. With 1 chart in text. [School of Trop. Med. & Hyg., Calcutta.]

The patient had suffered from rigors, intermittent fever and profuse sweats. Spleen palpable. *P. falciparum* found in blood. Quinine therapy failed to abolish fever. Liver became enlarged and tender, and bulging appeared in lower intercostal spaces. Total leucocytes 20,000. Four daily intramuscular injections of total alkaloids of *Holarrhena antidysenterica* had no effect on fever or liver condition. After a second injection of emetine hydrochlor. grain 1, temperature fell to and remained normal ; after 6 injections liver was normal to clinical examination. Total leucocytes 8,750. [It is regrettable that the *amoebic* nature of the hepatitis must be guessed only.]

H. M. H.

LEIBLY (Frank J.). **Fatal Emetin Poisoning, due to Cumulative Action, in Amoebic Dysentery.**—*Amer. Jl. Med. Sci.* 1930. June. Vol. 179. No. 6. pp. 834–839. With 1 chart in text. [16 refs.]

A woman, 21 years old, weighing about 100 pounds (37 kg.), suffering from diarrhoea and abdominal cramps, *E. histolytica* present in stools, was given (in 0·02 gm. doses each injection) intravenously 0·56 gm. of emetine hydrochlor. during 21 days, and again after 10 days interval, a further total of 0·72 gm. in 29 days, but this time given intramuscularly, 0·065 gm. each dose. She thus received 1·28 gm. or 0·034 gm. per kg. of body weight over a period of 60 days. The injections were stopped because of patient's general debility. Six days later death occurred from vasomotor collapse and heart failure, " brought on by the cumulative action of emetin."

At autopsy entire large intestine was examined for ulcers—only one small one (0·66 cm. diameter) was found just lateral to opening of ileum into caecum. Several scars as from healed ulcers. Veins throughout dilated ; heart to naked eye showed slight myocarditis [no report on histology of heart muscle]. By microscope liver showed parenchymatous degeneration

most marked about central veins. There was acute diffuse nephritis and acute parenchymatous degeneration of adrenals; acute hyperplasia of spleen. The emetine injections had failed to rid stools of pus and blood or of amoebae. The author records only "*Entamoeba histolytica*" and "amoebae present; few pus cells."

H. M. H.

HALAWANI (A.). **Experimental Study on the Resistance of *Entamoeba histolytica* to Emetine Hydrochloride *in vitro*.**—*Ann. Trop. Med. & Parasit.* 1930. July 8. Vol. 24. No. 2. pp. 273–288. [36 refs.] [School of Trop. Med., Liverpool.]

Full details are recorded of the author's experimental observations—they lead to the conclusion that:

"1. The resistance of *Entamoeba histolytica* to emetine hydrochloride can be raised by passing it systematically for a long period through graduated concentrations of that drug. 2. The amoeba undergoes a reduction in size after its passage over a long period through emetine hydrochloride. 3. The resistant amoeba retains its reproductive powers."

The author discusses the observations on resistant amoebiasis of various workers, and summarizes his most interesting and valuable research as follows:—

"The workers on this problem are, and will probably long remain divided in their theories as to the cause of resistant cases. The chief and most feasible of these theories advanced are:—

"1. An acquired resistance to emetine by *Entamoeba histolytica* after inefficient treatment. 2. Mechanical causes such as fibrosis around the diseased tissues preventing access of the drug via the blood. 3. Excystation of the resistant cysts in the bowels after cessation of treatment. 4. The hydrogen-ion-concentration plays a rôle in the efficacy of the drug. 5. Rapid excretion of the drug by the kidneys preventing it reaching the site of the lesion, as in the case of the cat. 6. A diminution of the curing properties of the drug during the cold weather is possibly associated with a change in the endocrine rate of metabolism leading to a loss of resistance on the part of the patient.

"Whatever may be the cause, or combination of causes of the occurrence of cases of amoebic dysentery resistant to emetine in spite of prolonged treatment, the results of my experiments lead me to conclude that one of the chief of these, is a tolerance to the drug gradually acquired by the amoebae. My culture of Mena Strain already showing some signs of resistance higher than the amoebae of the other three strains, was finally exalted through emetine tubes to a resistance four times that of untreated amoebae of the same strain, eight times that of the amoebae of V and G Strains, and five times that of the amoebae of Dobell and Laidlaw (1926). These results shed light on the interesting phenomenon of acquired drug tolerance already known to exist, to even a greater degree, among other parasitic members of the phylum protozoa."

H. M. H.

FAUST (Ernest Carroll). **Experimental Acute Amebic Colitis in Dogs.**—*Proc. Soc. Experim. Biol. & Med.* 1930. June. Vol. 27. No. 9. pp. 908–911. [9 refs.] [Dept. of Trop. Med., Tulane Univ., New Orleans.]

All evidence in the literature, as well as in the author's experience, preponderates in favour of belief that the amoeba in canine amoebic colitis is the same organism as *E. histolytica* of man. Since cysts are apparently not found in any type of the infection in dogs, the dog

is not likely to be a menace to man or to other dogs. The inference is, therefore, that the dog receives its infection from man. By injecting *per anum* into the distal ileum of the dog muco-sanguineous exudate from active amoebic colitis in dogs, the author produced infections corresponding to spontaneous dog-amoebiasis in 13 out of 14 dogs so injected—ranging in age from 2 months to 4 years. Incubation period as determined by presence of active *E. histolytica* in mucus withdrawn directly from caecum ranged from 1 to 23 days, with largest number on second day. Acute cases, probably complicated by bacteriaemia, die in about 2 weeks. Chronic cases and one spontaneous recovery were noted. No instance of a cyst passer was observed. The caecum is the primary seat of infection in the dog.

H. M. H.

HIYEDA (Kentaro). **Pathological-Anatomical Studies on Experimental Amoebiasis in Kittens.**—*Amer. Jl. Hyg.* 1930. Sept. Vol. 12. No. 2. pp. 401–423. With 5 text figs. [25 refs.] [School of Hyg. & Public Health, Johns Hopkins Univ., Baltimore.]

"1. Kittens were infected by rectal injections of *E. histolytica.*

"2. When *E. histolytica* is present in the stool, besides the changes indicated by white yellowish dotted areas representing aggregations of leucocytes in the dilated capillaries and the production of mucus, small excavations can be seen in the surface of the colon. These small excavations are follicular ulcers.

"3. After 24 hours in addition to the changes mentioned above a huge production of mucus and also of plasma occurs. *E. histolytica* are present in these masses, but have not yet entered the tissues.

"4. The changes of the early period mentioned above are a sign of general colitis due to the presence of amoebae in the lumen of the intestine, but not in the tissue itself.

"5. After 48 hours there are rough areas of different sizes in the mucous membrane. They consist of tissue debris, plasmatic exudates, mucus and amoebae. The upper layer of the epithelium in these rough areas is necrotic. The amoebae are found in the necrotic tissue and in the membrana propria, but they do not enter the deep part of Lieberkühn's glands.

"6. Later on these necrotic areas enlarge, on the one hand spreading superficially, and on the other going down through the membrana propria. The deeper part of the mucous membrane is invaded and destroyed by the amoebae in the following way; the epithelium of Lieberkühn's glands is attacked from the basal side, not from the lumina, and there results the exfoliation of epithelium from their basal membrane. *E. histolytica* intrude thus into the formed space. As a result of the disappearance of the epithelium the amoebae multiply and form a characteristic amoebic mass closely packed together.

"7. The pathological anatomical picture of amoebic colitis is quite characteristic. The following is a description, summarized and rather schematic. The amoebae in the intestinal tissue proceed approximately along the course of the connective or collagen fibres. In the superficial zone the amoebae have a tendency to spread parallel to the long axis of the intestine, because the fibres in this region run longitudinally. But it is clear that the superficial invasion cannot extend over a large area, as the supporting fibres run downward. From the superficial zone to the glandular part the amoebae move down within the membrana propria to the muscularis mucosa, since the fibres mostly run at right angles to the intestinal axis. In the deepest part of the membrana propria and in the submucosa the amoebae spread again parallel to the intestinal axis according to the course of the tissue.

" 8. These evidences are taken only from material from animals purposely killed. The material from animals which have been found dead after a certain period of infection sometimes suggests another mechanism, as for instance the invasion of Lieberkühn's glands by *E. histolytica* from the lumina of the intestine. My paper points out that these observations are not only made on insufficient material but are in contradiction to other biological facts known about *E. histolytica*.

" 9. The intestinal changes in the early period of colitis in experimental amoebiasis are not characteristic reactions due to the amoebae.

" 10. As to the probable exhaustion of the virulence of *E. histolytica* in the R.E.S. [Ringer-Egg-Serum] medium with starch, the present experiments are not sufficient in number to warrant a definite conclusion. The strain employed lost its virulence after three months and a half of cultivation, but was revived to a certain degree by retransference from the R.E.S. medium to the R.E.S. medium with starch."

On the question whether amoebiasis is a general disease or not, the author states that amoebae were found in the lymph and blood capillaries in the intestinal wall of his experimental kittens ; but the amoebae do not remain alive in the regional organs, for he has never found any in the liver, lung, kidney and spleen of the experimental kittens. The only change he found in the mesenteric glands was a sinus-catarrh, which happens also in bacillary dysentery and in some other intestinal disorders. He has seen very few amoebae in the lymph sinuses even when these were much degenerated.

H. M. H.

MARTIN (Dale L.). **The Lesions in Experimental Amebic Dysentery.**—*Arch. Pathology.* 1930. Sept. & Oct. Vol. 10. Nos. 3 & 4. pp. 349–385 ; 531–579. With 24 text figs. [133 refs.]

These are careful and valuable observations very fully set out and well illustrated.

In regard to culture mediums the author concludes that :—

" The original medium of Boeck and Drbohlav is best for the cultivation of *Endameba histolytica*. The substitution of a sugar-free cover fluid for Locke's solution, as suggested by Drbohlav, and the addition of rice starch, as recommended by Dobell and Laidlaw, constitute the most valuable modifications of these basic mediums. Although the effect is not striking, the addition of acriflavine to the supernatant fluid portion of the medium for the first one or two transplants may help to establish the continuous growth of cultivated amebas."

For the rest the paper must be consulted. The work is on the same lines as that of HIYEDA (above).

H. M. H.

SIMIC (T.). La résistance des amibes pathogènes à l'action du froid, de la chaleur et de la lumière solaire. [**The Resistance of Pathogenic Amoebae to Cold, Heat and Sunlight.**]—*Ann. Parasit. Humaine et Comparée.* 1930. July 1. Vol. 8. No. 3/4. pp. 225–230. [5 refs.] [Hyg. Inst., Skoplje, Yugoslavia.]

The author's experiments demonstrate that the vegetative forms of *E. histolytica, coli,* and *gingivalis* are resistant to low temperatures—0° C., the vegetative form of *E. histolytica* much more so than that of *E. coli* and *E. gingivalis* ; this biological character may be made use of in differential diagnosis between the first two amoebae. This resistance to cold increases directly with the number of passages in culture and most markedly

in the case of *E. histolytica*. *E. coli* and *E. histolytica* are sensitive to sun's rays, and degenerate much more quickly in the sun even at 22° C. than at 0° C. in the shade. The amoebae may be kept alive for a long time in stools by addition of Ringer's solution or normal saline solution. It is not cold but evaporation which kills the amoebae. Provided the stool be at once diluted with Ringer's, or with normal saline solution, diagnosis of *E. histolytica* can still be made, in the most unfavourable conditions, 8 to 10 hours after passage of stool.

H. M. H.

BEIJNEN (G. Koolemans). Over leveramoebiasis. [**Amoebiasis of the Liver.**]—*Nederl. Tijdschr. v. Geneesk.* 1930. Mar. 29. 74th Year. 1st Half. No. 13. pp. 1574–1583.

The article is a more or less verbatim transcription of a clinical lecture which enters very critically into the subject of differential diagnosis.

W. F. Harvey.

MACFADYEN (J. A.). **Clinical Observations on Amoebic Dysentery.**—*Jl. Med. Assoc. South Africa.* 1930. July 12. Vol. 4. No. 13. pp. 377–379.

The author states that in his private practice cases "proved to harbour the *E. histolytica*" were 4½ times more frequent than cases of bacillary dysentery [a complete reversal of the proportions for bacillary and amoebic dysentery actually proved everywhere else]. He does not state his proof except that "differential diagnosis is essentially microscopic."

H. M. H.

ABD-EL-AZIM (M.). **The Diagnosis of Amoebic Dysentery.**—*Jl. Egyptian Med. Assoc.* 1930. May. Vol. 13. No. 5. pp. 207–211. With 4 figs. on 1 plate. [4 refs.]

This is a brief recapitulation of the well-known points on which amoebic dysentery should be diagnosed: by the cellular picture of the stool, the morphological characters of *E. histolytica*, and the sigmoidoscopic appearance of the gut mucosa.

H. M. H.

BACILLARY DYSENTERY.

NELSON (Richard L.). **Sonne Dysentery. A Report of 32 Cases of Dysentery caused by** *Eberthella paradysenteriae* **Sonne.**—*Jl. Bacteriology.* 1930. Sept. Vol. 20. No. 3. pp. 183–201. [20 refs.] [Children's & Infants' Hosp., & Pediat. Dept., Harvard Med. School, Boston.] [Summary (with 2 tables not here reproduced) appears also in *Bulletin of Hygiene.*]

The author has encountered 32 cases of bacillary dysentery due to infection with the Sonne bacillus, 29 in children and infants, 3 in adults. Of these, 5 have been severe, the remainder mild. He gives six case histories, illustrating the milder and more serious forms of infection. He notes that the great majority of cases are not severe enough to cause any great concern, the illness being characterized by a short febrile attack, lasting from 24 to 48 hours, associated with diarrhoea setting in 24 to 48 hours after the onset of the fever and lasting from 2 to 6 days; mucus is common in the stools, blood is rare. Vomiting seldom occurs in these milder cases. A proportion of the cases are, however, of an altogether severer type, with frequent vomiting and

marked prostration. One such case in the present series was fatal. One case of the milder type was also fatal, but this occurred in a patient with uncompensated heart disease, and the dysenteric infection was probably only a contributory factor.

The biochemical and serological reactions of 28 strains of the Sonne bacillus isolated from these cases have been studied in some detail. The reactions of these strains have been compared with those of the *dispar* type described by ANDREWES, and of the metadysentery bacillus described by CASTELLANI. The results are set out in tables.

The author concludes that the metadysentery bacillus described by CASTELLANI is identical with Sonne's dysentery bacillus, while the *dispar* bacillus described by ANDREWES is an entirely different species.

W. W. C. Topley.

POWELL (A. T. W.). **Two apparently Unrelated Outbreaks of Bacillary Dysentery.**—*Brit. Med. Jl.* 1930. Aug. 2. pp. 173–175. [14 refs.]

The endemicity of dysentery in the British Isles is now fairly well established. Universal prevalence of the disease in late years appears only to be the consequence of recognition of this fact and more careful investigation of diarrhoeas. The outbreaks reported carefully in this article, together with the bacteriological and serological findings, have a special interest owing to the fact that they were produced by two different dysentery organisms, one of Flexner and the other of Sonne type. The Flexner outbreak provided the larger number of cases. In it the infection " bred true " throughout ; it resulted in two deaths and two relapses, and no source of infection either through water or milk supply could be discovered. A detailed enquiry gave no ground for the belief that there was any connexion between the Sonne outbreak and the Flexner although the common shallow well water supply of two of the families involved showed gross contamination.

W. F. Harvey.

RIDING (D.). **Acute Bacillary Dysentery in Khartoum Province, Sudan, with Special Reference to Bacteriophage Treatment : Bacteriological Investigation.**—*Jl. Hygiene.* 1930. Aug. Vol. 30. No. 3. pp. 387–401. With 4 charts. [1 ref.] [Wellcome Trop. Research Labs., Khartoum.]

Sixty cases of bacillary dysentery were observed during the 2 years of investigation and the causal bacilli were confirmed serologically in 43 of these—as Shiga, Flexner, or Flexner-Y organisms. After bacteriological identification the organism isolated was subjected to the action of bacteriophage *in vitro*. An organism which showed clearing of the broth suspension at the end of 24 hours and sterile agar subculture was described as readily bacteriophagable, one which showed incomplete clearing but formation of " plaques " in agar subcultures as bacteriophagable and one which showed neither of these characters as not bacteriophagable with the standard bacteriophage. Of the 60 bacilli isolated, only 12 failed to show evidence of bacteriophagy and only 2 out of the 43 strains which had been confirmed serologically. For treatment some of the cases were set apart as controls but, in order to avoid bias, the author was not aware of the type of treatment adopted until the investigation was complete. The administration of the

bacteriophage was entirely by the mouth. "There were no fatal cases in the series and no dramatic results due to the bacteriophage treatment were demonstrated." Factors which might possibly influence bacteriophagy *in vivo* were investigated. The author concludes that bacteriophage is probably quickly eliminated or destroyed and that the clinical course of acute bacillary dysentery is not altered by the oral administration of bacteriophage.

W. F. Harvey.

BURNET (F. M.), MCKIE (Margot) & WOOD (I. J.). **Investigations on Bacillary Dysentery in Infants, with Special Reference to Bacteriophage Phenomena.**—*Med. Jl. Australia.* 1930. July 19. 17th Year. Vol. 2. No. 3. pp. 71–78. With 1 text fig. [3 refs.] [Walter & Eliza Hall Inst. & Children's Hosp., Melbourne.]

A series of 21 cases of dysentery was investigated in infants of ages ranging from 3 to 22 months. Both the organisms and the phages obtained from these cases were classified. The most important of the phages ("N type") was a highly active one which did not readily provoke resistant variants and the appearance of this phage was found generally to be of favourable import for the patient. While it was evident that the effect of the presence or absence of the phage on the disease was negligible in the case of children over one year it seemed possible that the higher mortality in younger infants was correlated with absence of active phage in the faeces. Other investigations dealt with the relation of antigenic structure and the phage reactions of dysentery bacilli, change of type of infecting organism during the disease and a suggested method of typing dysentery strains by the use of a series of phages.

W. F. Harvey.

TAYLOR (J.), GREVAL (S. D. S.) & THANT (U.). **Bacteriophage in Bacillary Dysentery and Cholera.**—*Indian Jl. Med. Res.* 1930. July. Vol. 18. No. 1. pp. 117–136. [1 ref.]

Special care was taken to obtain suitable cases for effective treatment and this explains how it was that out of 878 dysentery cases in 1929 only 24 conformed to requirements. These requirements were, short duration of illness, characteristic cellular exudate and isolation of the causative organism. The object of the work was to determine what was the natural incidence of phage and what treatment with phage could effect in acute cases before severe ulceration and necrosis had taken place. All the cases, both treated and controls, received ordinary saline treatment and in no case was anti-dysentery serum administered. There were 20 controls to 26 cases of bacillary dysentery and the phage used was active to a dilution of 10^{-10}. The dose given was 2 cc. orally three times a day. The result was, in the words of the authors, that "In spite of the presence of phage of higher activity in the stools of treated cases as compared with controls no significant difference was observed in the progress of cases either in regard to effect on mortality or in shortening the period of the attack. A detailed examination of cases showed no relation between the incidence of phage either naturally developed or following therapeutic administration and the progress of the cases."

Much the same type of result was obtained with cholera cases, 19 of which were used as controls and 14 treated with bacteriophage of high activity. The mortalities were 53 per cent. for controls and 57 per cent. for cases treated with phage.

W. F. Harvey.

LONDON (J.). **Bacteriophage in its Clinical Aspect.**—*Indian Med. Gaz.* 1930. July. Vol. 65. No. 7. pp. 370–371. With 1 chart in text. [2 refs.]

The author describes the results obtained from oral administration of bacteriophage in bacillary dysentery. Of 141 cases treated, 129 were cured and 12 died. In this total, 108 received no other treatment than bacteriophage and were discharged cured. The remainder did not respond so well.

W. F. Harvey.

VIOLLE (H.). De la pathogénie de la dysenterie bacillaire. [**Pathogeny of Bacillary Dysentery.**]—*Bull. Acad. Méd.* 1930. July 8. Year 94. 3rd Ser. Vol. 104. No. 27. pp. 59–62. [6 refs.]

The author briefly reviews in a general statement the results of his experiments on the immunization of rabbits against Shiga dysentery bacillus. Live Shiga bacilli introduced by the subcutaneous, intramuscular, intravenous, or oral route into the rabbit always select the small intestine ; they settle and multiply there. The immunizing dose of living Shiga bacillus directly introduced into the small intestine is too near the lethal dose to provide an effective means of immunization. A dose of living Shiga bacilli injected (after laparotomy) through the external wall of the rabbit's small intestine into the intestinal submucosa procured perfect immunity from doses of living Shiga bacilli which proved lethal to control animals, and this immunity held whether the lethal dose was introduced intravenously or directly into intestine. The immunized rabbit's serum, however, showed *in vitro* neither specific agglutinins nor fixation of complement. The dose of living Shiga bacillus, which was non-lethal but immunizing when injected into the submucosa of the small intestine of the rabbit, was lethal to the rabbit if injected intravenously. Experimental observations showed that intestinal mucosa *in vitro* has the power of neutralizing products of Shiga bacillus and *in vivo* experiments appeared to confirm this. In the normal person the syndrome of dysentery would not appear ; there must be exceptional conditions for it to develop.

H. M. H.

ALIVISATOS (G. P.). Die perorale Immunisierung gegen Ruhr. [**Peroral Immunization against Dysentery.**]—*Immunität, Allergie u. Infektionskr.* Munich. 1929–30. Vol. 2. No. 1/2. pp. 37–52.

A careful account is given at considerable length of the basis and advantages of peroral immunization against dysentery. The author's own statistics showed an incidence of 3 cases (0·8 per cent.) among 373 treated and 96 cases (23·2 per cent.) among the 417 control untreated persons. Much larger doses than the usual total of 0·15 gm. were given. They were 0·2 to 0·24 gm. (total) of Shiga-Kruse bacilli, killed at 55° C.

and formolized at 0·15 per cent., on 5 to 6 days, often with an interval of 5 to 6 days between the 3rd and 4th doses. These amounts were well borne by adults but should be reduced to half for children under 12 years of age. It is necessary to realize that no cross immunity is obtainable between Shiga-Kruse and paradysentery bacillary infections. Accordingly, the organism concerned in an epidemic must be that which is used in immunization. Immunization will last for at least a dysentery season.

W. F. Harvey.

CHASKINA-MUNDER (G.). Dysenteriebacillen im Vaginalsekret junger Mädchen.—*Ztschr. f. Kinderheilk.* 1929. Vol. 48. pp. 690–693. [6 refs.]

——. [**Dysentery Bacilli in the Vaginal Secretion of Girls.**]—*Trop. Med. & Veterin.* Moscow. 1930. Vol. 8. No. 2. pp. 47–49. [6 refs.] [In Russian.]

The author examined 24 girls with symptoms of non-specific vulvo-vaginitis. From the vagina of two of these Flexner's bacilli were isolated. Their nature was confirmed both by cultural and serological reactions and by inoculation of rabbits. In one case the blood showed the agglutination reaction. It is concluded that the condition in both cases was due to the micro-organism in question. The examination of the faeces of these children was negative, nor were dysenteric symptoms present.

C. A. Hoare.

BEZEMER (F.). *Bacterium dysenteriae* Shiga-Kruse in het bloed tijdens het leven. [*Bacterium dysenteriae* **Shiga-Kruse in the Blood during Lifetime.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1930. June 1. Vol. 70. No. 6. pp. 551–556. [22 refs.]

From a bile culture of blood of a European patient of 39, as well as from his faeces, the Shiga-Kruse bacillus was isolated. The agglutination titre of the patient's serum against the strain was 1 : 100, that against pseudo-dysentery bacillus 1 : 200. The patient recovered.

Some quotations from the literature serve to show the rarity of this finding.

W. J. Bais.

CERRUTI (Carlo F.). **The Metadysentery Bacilli (Castellani).**—*Jl. Trop. Med. & Hyg.* 1930. July 15. Vol. 33. No. 14. pp. 207–210. [29 refs.] [Royal Inst. of Hyg., Univ., Turin.]

The author considers that the classification of dysentery bacilli devised by CASTELLANI is practical and scientific. He discusses the generic position of the metadysentery bacilli and gives the principal species with their reactions.

W. F. Harvey.

STETTNER (Ernst). Ueber die Behandlung der Bazillenruhr im Kindesalter. [**Treatment of Bacillary Dysentery in Children.**]—*Immunität. Allergie u. Infektionskr.* Munich. 1928–29. Vol. 1. pp. 367–377. [11 refs.] [Univ. Children's Clinic, Erlangen.]

In a discursive statement of his own experience the author stresses the importance of dietary treatment, in which fluids must be liberally supplied.

Among drugs he claims yatren, by mouth and in clysma, as giving the best results.

H. M. H.

NAKAJIMA (Gishiro). **On Cystin Reduction by Dysentery Group.**—*Densenbyo Gakkaishi (Jl. Infect. Dis. Soc.).* 1929. Aug. Vol. 3. No. 11. [Summarized in *Japan Med. World.* 1930. Jan. 15. Vol. 10. No. 1. pp. 16–17.]

The Sonne strain of dysentery is differentiated from other members of the dysentery group by its reduction of cystin in an oryzanin non-protein medium with production of hydrogen sulphide. In this it resembles typhoid, paratyphoid and coli organisms.

W. F. Harvey.

MIXED AND UNCLASSED DYSENTERY.

DUNBAR (L.) & STEPHENS (E. D.). **Notes on the Dysenteries of the Madras District.**—*Indian Jl. Med. Res.* 1930. July. Vol. 18. No. 1. pp. 149–163. With 1 chart in text. [5 refs.]

Amoebic dysentery has long been regarded as the chief dysentery in the Madras district. In actual fact bacillary outnumbers amoebic dysentery by about five to one. The commonest causal organism is a bacillus of Flexner type. The bacillary dysentery as seen by the authors was mild but present all the year round. Healthy carriers are rare—0·34 per cent. Indian carriers of *E. histolytica* cysts are common, at least 21·9 per cent., and the British soldier carries *E. histolytica* cysts at approximately the same rate ; it is conceivable that the *E. histolytica* of this district is non-pathogenic. It is equally conceivable that amoebic dysentery is always secondary to bacillary.

H. M. H.

PERRY (H. Marrian). **Observations on Dysenteric Infections in Egypt.**—*Jl. Egyptian Med. Assoc.* 1930. Apr. Vol. 13. No. 4. pp. 93–105.

Both in adults and in children 75 per cent. of the cases are bacillary, and 15 per cent. amoebic : 50 per cent. of bacillary cases are due to one or other Flexner bacilli ; the more severe type, the Shiga bacillus infection, accounts for 10 per cent., and the Sonne bacillus is causative agent in 15 per cent. In a small number of cases an acute Flexner group bacillus infection was found superimposed on *E. histolytica* infection.

The above results are based on examination of over 800 cases of acute dysentery and can be accepted as accurate classification of the type of disease prevalent in Egypt.

A provisional diagnosis of the type of dysentery can be made on the character of the cellular exudate.

H. M. H.

SHAWKI (I.). **Dysenteries in Children in Egypt.**—*C. R. Congrès Internat. de Méd. Trop. et d' Hyg. Le Caire, Egypte, Décembre, 1928.* Vol. 2. pp. 929–934. [12 refs.]

The author reports on 98 cases of dysentery in children 6 months to 8 years old in which there had been thorough microscopical and cultural

investigation of stools, the bacteria isolated being further tested against known immune sera. Amoebae or lamblia were found in 12 per cent. Of 76 per cent. in which bacteria were isolated Flexner or Y bacillus were found in 52 per cent. ; Shiga in 23·5 ; and Morgan No. 1 bacillus in 20 per cent. No pathogenic agent in 12 per cent.

H. M. H.

BIGGAM (A. G.). **The Sigmoidoscope as an Aid in the Diagnosis of Dysenteric Conditions in Egypt.**—*Jl. Egyptian Med. Assoc.* 1930. May. Vol. 13. No. 5. pp. 149–167.

——. **The Diagnosis of Dysenteries.**—*Jl. Roy. Army Med. Corps.* 1930. July. Vol. 55. No. 1. pp. 16–24.

—— & ARAFA (M. A.). **The Sigmoidoscope as an Aid in the Diagnosis of Dysenteric Conditions in Egypt.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1930. Aug. 8. Vol. 24. No. 2. pp. 187–200. With 12 coloured figs. on 3 plates.

The author has found preliminary dieting and lavage to clear the bowel of faecal material better than attempts at cleansing by lavage and general swabbing through the sigmoidoscope. The instrument is passed for three inches, obturator removed, and further passage carried out under direct vision aided by gentle air inflation. Even in extensively ulcerated guts this gentle air inflation has caused no pain nor been harmful. Mucus can be removed by swabbing so as to get clear view of submucosa and determine if ulcers, as they often do, underlie patches of slimy mucus. The magnifying glass fitted to instrument is necessary for finding the smallest ulcers.

Amoebic, bacillary and bilharzial dysentery are then considered in turn ; the appearances are carefully described and illustrated by good coloured plates (Biggam & Arafa).

H. M. H.

SMYLY (H. Jocelyn). **The Diagnosis and Treatment of Chronic Dysentery.**—*Trans. Roy. Soc. Trop. Med. & Hyg.* 1930. June 30. Vol. 24. No. 1. pp. 39–66. With 12 coloured figs. on 1 plate. [17 refs.] [Peiping Union Med. College, Peking.]

A full and instructive record, well illustrated, of careful observations in Peking, of great clinical value. They emphasize the importance for accurate diagnosis of sigmoidoscopy. They show that a higher number of positive results is given if culture be made at once of swabs or scrapings of colon ulcers obtained directly through sigmoidoscope, and that it is misleading to diagnose merely on microscopic findings in material obtained by swabbing or scraping of gut, for in some cases positive cultures of *Bacterium dysenteriae* were obtained from curette-material which also showed microscopically many motile *E. histolytica* ; and in several other cases *E. histolytica* cysts were found in the stools, where the colon ulcers failed to give amoebae, but gave positive cultures of *Bact. dysenteriae.* In Peking where 30 per cent. (KESSEL and SVENSSON 1924) of community are *E. histolytica* carriers it was to be expected that a large number of bacillary dysentery patients would be *E. histolytica* carriers. The author's observations form important additional evidence in support of the contention that " chronic ulcerative colitis " is " chronic bacillary dysentery."

Fifty-six Cases of Bacillary Dysentery showing Duration of Disease when Positive Cultures were obtained.

Organism.*	Months.												Totals, 1 to 12 months.	Years.							Totals over 1 year.
	1	2	3	4	5	6	7	8	9	10	11	12		1	2	3	4	5	6	10	
M.F. ...	1	1	1	2		1		1	1	2	1	1	12	5	2	1	3	2			13
S.		2	1			2		1					6	3	3	3				1	10
M.F.+S. ...				1	1	1	1						4	1				1			2
M.F.+E.h.		1	1	1		1		1					5								
S.+E.h. ...						1	1						2								
M.F.+S. +E.h. ...															1				1		2
Totals ...	1	4	3	4	1	6	2	3	1	2	1	1	29	9	6	4	3	3	1	1	27

* M.F. = *Bact. dysenteriae*, Mannite-fermenting group.
S. = *Bact. dysenteriae*, Shiga type.
E.h. = *Entamoeba histolytica.*

H. M. H.

WALKER (J.). Quelques observations bactériologiques et épidémiologiques au sujet des dysenteries et entérites du Katanga. [**Dysentery and Enteritis in Katanga : Bacteriology and Epidemiology.**] —*Ann. Soc. Belge de Méd. Trop.* 1930. June 30. Vol. 10. No. 2. pp. 181–212.

The author's observations lead him to conclude :—

1. The dysenteries and dysenteric enteritis of the Katanga are divisible for the most part into two categories—bacillary dysenteries and alimentary intoxications.

2. Most of the bacillary dysentery was caused by a Shiga bacterium, or by other bacteria belonging to the genus Shigella (Castellani and Chalmers). In some cases organisms of the genus Lankoïdes were isolated.

3. The alimentary intoxications were due to Proteus bacillus, Enteroides and Balkanella (group Ebertho-Aerogenes of Castellani and Chalmers), and occasionally probably to other saprophytes such as the Faecalis alcaligenes bacillus, Pyocyaneus, or perhaps even to toxic strains of the colon bacillus.

4. In a minority of cases were found organisms of the Salmonella (Aertryck) group, Morgan's bacillus, and Castellani's Asiaticus group. Certain serological reactions indicated that these organisms were present oftener than could be established by the stool examinations.

5. Bacillary dysentery prevails in the cold season in epidemic outbreaks, always shows the classical features of dysentery, is mild and yields readily to proper treatment. Mortality 13 per cent., but the fatal cases were complicated by other affections.

6. The alimentary intoxications prevail in the hot season among the poor and ill-nourished natives ; are not epidemic ; often conjoined with or succeeding some other bacillary or parasitic infection. Polymorphic clinical features ; difficult to treat ; mortality 20 per cent.

7. The dysentery bacilli isolated were in general agglutinated by an anti-Shiga serum and by the patients' own serum. The organisms of the

alimentary intoxications rarely formed agglutinins; sometimes specific agglutination appeared in the course of the illness. Agglutination of colon bacillus by the patients' serum was frequent. H. M. H.

MENON (T. Bhaskara). **The Pathology of Chronic Colitis in the Tropics.** —*Indian Jl. Med. Res.* 1930. July. Vol. 18. No. 1. pp. 137–141. With 7 coloured figs. on 5 plates. [6 refs.] [Med. College, Madras.]

In a valuable paper, well illustrated, the author describes the different types of dysenteric lesions observed in 800 autopsies. Histologically "stercoral" ulcers are indistinguishable from chronic dysenteric ulcers; 60 per cent. of fatal cases of "chronic" colitis or "chronic diarrhoea" of indefinite origin show morphologically strong resemblance to the lesions of chronic dysentery; some cases showed an atrophied state of all the coats of the large and small intestine, the exact cause of which was not determined. H. M. H.

CHOPRA (R. N.). *Plantago ovata*—**Ispaghul—in Chronic Diarrhoeas and Dysenteries.**—*Indian Med. Gaz.* 1930. Aug. Vol. 65. No. 8. pp. 428–433. [4 refs.] [School of Trop. Med. & Hyg., Calcutta.]

In the last 15 years the author has given very extensive clinical trials to the seeds of *Plantago ovata*, the chemical composition, pharmacological action, and therapeutic uses of which are described in this paper. The author has used it in: (1) chronic bacillary dysentery; (2) chronic amoebic dysentery; (3) chronic constipation, with auto-intoxication due to other causes; (4) "Hill diarrhoea"; (5) chronic diarrhoea in children. In all these conditions he claims the seeds of *P. ovata* to be very beneficial. A glucoside, *Aucubin*, has been found in the seeds, but is physiologically inactive. The tannins present in appreciable quantities have little action on entamoeba or bacterium. Action of *P. ovata* is therefore mechanical; due to the large amount of mucilage in superficial layers of the seeds. This mucilage is shown not to be acted on by the digestive enzymes, and therefore passes unchanged through the small intestine; it lines the mucosa of the gut and as demulcent is protective and sedative. In the large gut the bacteria have been shown to have little or no action on the mucilage—practically the whole of it is passed out unchanged 12 to 24 hours after administration. It coats the inflamed ulcerated mucosa and protects it from irritation of fluids and gases. This enables the lesions to heal quickly. Toxins in gut are adsorbed by the gel and their absorption into the system prevented. The seeds are taken in large quantities, and as they swell up in contact with water they increase the bulk of gut contents and thus relieve constipation by mechanically stimulating gut peristalsis. H. M. H.

GHEORGHIU (I.). Contribution à l'étude d'une épidémie de gastro-entérite dysentériforme. [**Study of an Epidemic of Dysenteriform Gastro-Enteritis.**]—*Ann. Inst. Pasteur.* 1930. June. Vol. 44. No. 6. pp. 711–718. With 2 text figs. [2 refs.] [Bact. Lab., Faculty of Med., Jassy.]

Entirely inconclusive observations on 186 cases, for no pathogenic agent was demonstrated by culture or serological tests. H. M. H.

DE ASSUMPÇÃO (Lucas). Notas sobre a fermentação tardia e phenomeno de "caméléonage" com bacillos dysentericos.—*Bol. Inst. Hyg. de São Paulo.* 1929. No. 42. 37 pp. English summary pp. 37–38.

CASTELLANI (Aldo). La colite cronica da bacilli metadissenterici (metadissenteria cronica).—*Ann. di Med. Nav. e Colon.* 1930. Jan.–Feb. Year 36. Vol. 1. No. 1/2. pp. 1–9. [14 refs.]

CASTEX (M. R.), POLETTI (R. A.) & LÓPEZ GARCÍA (A.). El sondaje duodenal como coadyuvante en el diagnostico de la hepatitis amebiana supurada.—*Arch. Argentinos Enferm. Aparato Digest. y Nutric.* Buenos Aires. 1930. Aug.–Sept. Vol. 5. No. 6. pp. 904–906.

FARMAKIDIS (C.). A propos d'un cas de tétanie au cours de la dysenterie amibienne.—*Arch. des Malad. de l'Appareil Digestif.* 1930. June. Vol. 20. No. 6. pp. 669–674.

GESSNER (Otto). Schlussbemerkung zu der Erwiderung von O. Schaumann auf meine Bemerkung in diesem Archiv, Bd.33, S.277 ff.—*Arch. f. Schiffs- u. Trop.-Hyg.* 1929. Sept. Vol. 33. No. 9. p. 492. [1 ref.]

HEYD (C. G.) & SHEPLAR (A. E.). Amebic Dysentery: Seven Cases.—*Amer. Jl. Surgery.* 1930. Jan. Vol. 8. p. 54. [Summarized in *Jl. Amer. Med. Assoc.* 1930. Mar. 22. Vol. 94. No. 12. p. 895.]

HOSHI (Naotoshi). Effect of Various Absorbents upon the Toxins of the Dysentery Bacillus.—*Jl. Oriental Med.* 1930. Jan. Vol. 12. No. 1. pp. 3–5. [General Hosp., Dairen.]

MANDRY (O. Costa). Diarrhea and Enteritis in Porto Rico. Bacteriological and Parasitological Study of Infantile Diarrheas.—*Bol. Asoc. Med. de Puerto Rico.* 1930. July. Vol. 22. No. 178. pp. 83–91. With 1 chart. [16 refs.]

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DA SILVA-MELLO (A.). Die chronische Amöbeninfektion des Darmes und ein für ihre Diagnose bedeutsames Symptom.—*Arch. f. Schiffs- u. Trop.-Hyg.* 1930. Aug. Vol. 34. No. 8. pp. 411–422.

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YELLOW FEVER.

JORGE (Ricardo). La fièvre jaune et son extinction à Rio de Janeiro. [**Yellow Fever and its Extinction at Rio de Janeiro.**]—*Bull. Office Internat. d' Hyg. Publique.* 1930. Aug. Vol. 22. No. 8. pp. 1481–1493. With 2 text figs. [8 refs.]

An interesting and instructive summary of certain aspects of the epidemiology of yellow fever with special reference to the Rio epidemic which came to an end in July 1929. With regard to the incidence of the disease 80 per cent. of the cases occurred in persons more than 15 years old, and 76 per cent. were males. Ninety-two per cent. were whites, 64 per cent. foreigners, and 60 per cent. of the patients had less than 5 years' residence. The fatality of various groups of the population varied between 55 and 67 per cent., with a mean of 59·6, except in the case of negroes ; there were only 6 cases with 1 death in this group, and although these figures are very small, there is additional evidence that the negroes are relatively resistant. An examination of the statistics shows that in the case of mulattos, who numbered only 7 per cent. of the total number of cases, the fatality was 67 per cent. as compared with 66 per cent. among whites. It is not easy to explain the relative immunity of mulattos to this infection, for it is evident that once having become infected the disease is quite as fatal to them. The supposed resistance of negroes requires further investigation. Attention is called to the importance of non-apparent cases in which the nature of the disease cannot be detected by clinical examination and in West Africa the work of the Rockefeller Commission (see below, p. 283) has shown the existence of endemic areas of yellow fever even in regions where cases of the disease have not been observed.

The author then discusses the possible significance of monkeys as a reservoir of the virus and the results of recent infection experiments with various species, and concludes with reference to work on the nature of the virus and on yellow fever vaccine. As the author aptly remarks, " with an efficient vaccine yellow fever prophylaxis will be completely armed."

E. Hindle.

PEÑA CHAVARRIA (Antonio), SERPA (Roberto) & BEVIER (George). **Yellow Fever in Colombia with Special Reference to the Epidemic in Socorro in 1929.**—*Jl. Preventive Med.* 1930. Nov. Vol. 4. No. 6. pp. 417–457. With 5 text figs. [44 refs.]

A general historical account of yellow fever epidemics in Colombia since the first known outbreak in 1509. It is stated that the disease probably did not occur in the interior until about 1830, when it extended up the Magdalena River and from Maracaibo up to the Catatumba Zulia Valley. Since 1900 Santander seems to have been the centre of infection with severe epidemics in 1910, 1923 and 1929. During January and February, 1929, an epidemic, locally supposed to have been influenza, occurred in Guadalupe, Santander ; possibly some of these cases were yellow fever. From April to July, 1929, in the neighbouring town of Socorro, there were 150 cases of yellow fever with 34 deaths. Antimosquito measures soon brought the epidemic under control, but it is curious that the disease apparently did not extend to other towns, in some of which *Aedes aegypti* was more common than in Socorro. Clinically there were two forms of the disease :—

"One was mild or abortive, beginning suddenly with a chill, severe and persistent pain in the back, headache, pains in muscles and joints, vomiting, fever, bloated face, injected conjunctivae and mucous membranes, an increased pulse rate, frequently a mild icterus, scanty and albuminous urine, and a sudden reaction for the better at the end of 24 to 36 hours, followed by convalescence. The other form began in much the same way, perhaps with more marked symptoms, accompanied by restlessness and a sense of anxiety. A slight remission, corresponding to the onset of convalescence in the mild type, was followed by a prompt return of the temperature to 38° or 39° C., slow pulse rate, increased icterus, and decided hemorrhagic tendencies such as bleeding gums, epistaxis, melena, black vomit, and even pui puric infiltrations of the skin. The urine, which was scanty or suppressed entirely, contained great quantities of albumin. Shortly after death the cadaver took on a characteristic yellow color."

The pathological and histological findings were typical and monkeys inoculated with the serum of recovered cases were protected against a subsequent injection of yellow fever virus. Many positive results were also obtained with the sera of patients who had suffered from yellow fever in Bucaramanga in 1910 and 1923, and in Guadalupe in 1929. The Santander region where these two towns and also Socorro are situated is so isolated by high mountains that the introduction of infected persons or mosquitoes is considered very unlikely. The recent epidemics suggest that the disease may be endemic in this region, maintained by mild unrecognized cases. E. H.

GAITAN (Luis). La fièvre jaune au Guatémala. [**Yellow Fever in Guatemala.**]—*Bol. Sanit. de Guatemala.* 1929. Oct.–Dec. Vol. 1. No. 2. p. 65. [Summarized in *Bull. Office Internat. d' Hyg. Publique.* 1930. Dec. Vol. 22. No. 12. p. 2308.]

The author considers that the disease is endemic along the sea coast of Caraiba and that there is a constant danger of epidemics, especially in view of the large number of Aedes in the coastal region of the Republic. This century epidemics have occurred in 1903, in 1918, when there were 609 cases with a mortality of 42 per cent., and in 1920 and 1921 on the borders of Honduras ; since then no cases seem to have been recorded. E. H.

LUTZ (Adolpho). [In Portuguese & German.] Reminiscencias da febre amarella no Estado de São Paulo. Erinnerungen aus der Gelbfieberzeit im Staate São Paulo. [**Reminiscences of Yellow Fever in São Paulo.**]—*Mem. Inst. Oswaldo Cruz.* 1930. Oct. Vol. 24. No. 3. In Portuguese pp. 127–142. In German pp. 143–160.

A very interesting account of the author's long personal experiences of this disease extending back to the eighties of last century. Many aspects of yellow fever are referred to and the paper should be read in its entirety by all those interested in the subject. E. H.

LAMY (P.). L'épidémie de fièvre jaune et de peste en 1899 à la Côte d'Ivoire. [**The Epidemic of Yellow Fever and Plague in 1899 on the Ivory Coast.**]—*Bull. Soc. Path. Exot.* 1930. Nov. 12. Vol. 23. No. 9. pp. 891–898.

A dramatic account of an epidemic of yellow fever during April and May, 1899, at Bassam on the Ivory Coast. The author's two medical

colleagues died of yellow fever, and the 33 resident white population all became infected and 30 died of this disease during these two months. The author remarks that after considerable experience at the Front during the Great War, including many important engagements, he has never experienced such a feeling of hopeless terror, approaching panic, as during his residence at Bassam, for at that time the method of infection was unknown. Eventually the author himself became infected; he describes his symptoms with graphic details, and the anxious interest with which he followed the amount of albumin in his urine.

The author submitted an official report of this epidemic, which was evidently suppressed as there was no mention of it in any of the medical journals of the time. Moreover, on returning to Paris, he made a verbal report as to the nature of the infection, but the administrative authorities refused to admit the existence of yellow fever in that region since it would have interfered with commerce and discouraged future colonists.

E. H.

BEEUWKES (Henry), BAUER (J. H.) & MAHAFFY (A. F.). **Yellow Fever Endemicity in West Africa, with Special Reference to Protection Tests.**—*Amer. Jl. Trop. Med.* 1930. Sept. Vol. 10. No. 5. pp. 305–333. With 2 maps in text. [4 refs.] [West African Yellow Fever Commission, Internat. Health Division, Rockefeller Foundation, Lagos.]

A most important article on the epidemiology of yellow fever in West Africa. After an interesting discussion of the various regions of West Africa, the authors bring evidence to show that a densely populated area in Southwestern Nigeria, including Ibadan and many other large towns, is a definite endemic centre of the disease.

It is well known that the blood of persons who have recovered from yellow fever will protect rhesus monkeys against the infection and particulars are given of 3 cases in which the serum protected monkeys after intervals of 24 to 27 years since the attack of the disease. Consequently, protection tests were carried out with the sera of natives taken at random in several towns.

As a routine method 5 cc. of serum was injected intraperitoneally into each monkey, and the virus was usually inoculated 3 to 24 hours later. Two monkeys were used for each experiment since these animals sometimes die from intercurrent infections and occasionally are refractory.

The results of protection tests with the sera of natives from various West African towns is summarized in the following table.

It will be noticed that among 125 sera collected at random from persons living in Ibadan and Ilorin, in the region considered endemic, 30·4 per cent. protected duplicate monkeys, and in Ife, where an epidemic had recently occurred, 17 out of 25 sera, or 68 per cent., protected two animals. In contrast with these results, in Northern Nigeria out of 90 examined only one person was found to be positive. Twenty-four per cent. of the sera of children between the ages of 4 and 5 years taken at random in Ibadan, and a similar percentage of sera from children at Ilorin between 4 and 7 years, protected duplicate monkeys. It seems, therefore, that at least one of every four young children in these cities has had an attack of yellow fever. Moreover, the authors bring forward strong evidence in support of the view that the actual number of children who have had the disease is probably greater than these figures suggest.

Towns.	Total No. of specimens tested.	No. of sera which protected both monkeys.	No. of sera which showed no protection.	No. of sera with which only one monkey died.
1. Southwestern Nigeria :				
Ibadan	100	32	50	18
Ilorin	25	6	10	9
Total	125	38	60	27
2. Northern Nigeria :				
Bauchi	36	0	35	1
Zaria	29	0	27	2
Kano	25	1	21	3
Total	90	1	83	6
3. Ife, after an epidemic of yellow fever ...	25	17	6	2
4. Freetown. No yellow fever reported since 1910	34	1	30	3

During the extensive epidemics studied on the Gold Coast, a few babes-in-arms with scleral icterus were observed, but no child under 5 years presented a clinical picture justifying the diagnosis of yellow fever.

With regard to the experimental details it was found that not more than 6 per cent. of normal rhesus monkeys survived the injection of 0·1 cc. of virulent blood taken at the onset of fever. Dilution experiments, using 10 to 25 per cent. normal serum as a diluent, have shown that amounts of between 0·0000001 and 0·000000001 cc. of infected blood have frequently proved fatal, and in these protection tests the amount of blood used represented approximately 1,000,000 lethal doses.

The amount of immune serum is of importance, but 1 to 2 cc. were repeatedly found to protect against large doses of virus, although on one occasion 2 cc. of this same immune serum when tested in duplicate monkeys protected one and not the other.

[It is unnecessary to draw attention to the great importance of these observations on the prevalence of yellow fever in the various regions selected by the authors. The results in Ibadan and Ilorin show that this region is a great endemic centre of the disease, and support the view advanced by Sir Rubert Boyce 20 years previously, that the West African native is as saturated with yellow fever as he is with malaria.]

E. H.

Hudson (N. Paul) & Kitchen (S. F.). **Postepidemic Diagnosis of Yellow Fever by the Passive Immunity Test.**—*Jl. Preventive Med.* 1930. Nov. Vol. 4. No. 6. pp. 459–462. [10 refs.] [Yellow Fever Lab., Internat. Health Division, Rockefeller Foundation, New York City.]

This paper contains details of the passive immunity tests made with the blood of 13 recovered cases of suspected yellow fever from Bucara-

manga, Guadalupe, and Socorro. Two of the Guadalupe cases and one Bucaramanga case were negative, but all the others were positive. A West African strain of virus (Asibi) was used in these experiments, which therefore show that yellow fever in Colombia is immunologically identical with the disease in West Africa.

E. H.

HINDLE (Edward). **The Transmission of Yellow Fever.**—*Lancet.* 1930. Oct. 18. pp. 835–842. [21 refs.] [Wellcome Bureau of Scientific Research, London.]

This paper contains records of numerous experiments on the transmission of yellow fever carried out during the past two and a half years at the Wellcome Bureau of Scientific Research. Four strains of virus were used, three African and one Brazilian, but no marked difference could be found between them. Vaccination or other methods of immunization against one strain also protected against all other strains and these results support the now generally accepted view as to the identity of the African and American strains.

Direct transmission. The course of infection in man and monkeys is next considered and it is shown that whilst during the incubation period in both the blood is presumably infected, once fever has developed the blood generally ceases to be infective by the fourth day and often earlier. Monkeys commonly die before the fourth day of fever and their tissues are generally infective at death, but human patients rarely die before the 5th or 6th day of fever and consequently their blood has ceased to be infective before death. This would seem to offer some explanation of the large number of cases of laboratory infection that have occurred since monkeys were used, as compared with the absence of any record of disease being acquired by conducting post-mortem examinations on the human cases of yellow fever. The method of infection in these laboratory cases seems to have been by passage of the virus through the intact skin and records are given of recent infections among the author's laboratory attendants, and also in hospital technicians. [For full details of these cases see below, LOW & FAIRLEY, p. 290.] Two of these persons could have acquired the infection only when performing a blood examination on human yellow fever blood and in both instances developed the disease exactly 10 days after coming in contact with the infected material. The blood of one of these patients was found to be infective to monkeys on the fourth day of fever, in spite of the injection of 30 cc. of immune serum on the third day. These laboratory infections were acquired from a Brazilian virus that had been passaged through monkeys for a year and included a mild case and severe cases, one fatal; it is evident, therefore, that passage in monkeys does not diminish the virulence of the virus towards human subjects.

Transmission by mosquitoes. It was found that the incubation period elapsing before the mosquito becomes infective might be as short as 9 days at a constant temperature of 28° C., or indefinitely prolonged if the mosquitoes were kept at low temperatures. Thus a mosquito that has ingested virus, if kept at a temperature of 10° to 15° C., does not become infective, but if after several weeks the temperature is subsequently raised to 28° C. it becomes infective. On the other hand, a mosquito that has become infective retains its infectivity by bite even

if kept at temperatures as low as 10° to 15° C. A mosquito may become infective if kept at temperatures as low as 18° C. but the incubation period is very prolonged. Concerning the proportion of *Aedes aegypti* becoming infected after feeding on a yellow fever monkey, in one experiment, out of 11 mosquitoes 5 (or 6) became infective, and in the remainder the infection died out. These mosquitoes were kept at a temperature of 28° C. A fatal infection was produced by the bite of a mosquito 118 days after it had been infected, and there seems no doubt that once infected a mosquito generally remains infective for the duration of its life. The inoculation of eggs laid by infected parents, and also of larvae, pupae and adults developing from such eggs, gave uniformly negative results, and it seems almost certain that the infection is not transmitted to the offspring of an infected mosquito. With reference to the passage of the virus from one mosquito to another during copulation [see ARAGÃO, this *Bulletin*, Vol. 26, p. 1001], the author found that this was due to a surface contamination of the insects' bodies with infected faeces and was removable by washing. Consequently these cases are considered to have no bearing on the persistance of the disease in nature. Experiments with immune serum and virus showed that the loss of infectivity to mosquitoes, which generally occurs after one to three days of fever, is due to the development of immune bodies in the blood and not to the disappearance of virus. Mosquitoes fed on a mixture of virus and immune serum failed to become infected and yet these mixtures were infective when inoculated into monkeys. In yellow fever cases the blood ceases to be infective to mosquitoes as soon as the concentration of immune bodies reaches a certain level, but the virus does not disappear from the blood till much later. The ingestion of immune serum was found to have no effect on the infection once it was established in the mosquito, and infected mosquitoes remained infective after having been fed on immune blood.

E. H.

FOWLER (James K.). **Recent Work on Yellow Fever.** [Correspondence.]—*Brit. Med. Jl.* 1931. Jan. 10. p. 73.

An interesting letter with special reference to the Yellow Fever Commission (West Africa), of which the writer was chairman. Apparently all the reports of this Commission not distributed at the time of publication were lost, and have only recently been found. Copies may now be obtained on application to Dr. A. T. STANTON, or to the Crown Agents for the Colonies, Millbank. It is unfortunate that the urgent representations made in these reports did not receive more attention at the time, for the likelihood of monkeys and especially "howlers" being susceptible to yellow fever was embodied in their scheme for further research.

The numerous laboratory infections which have recently occurred in this country and elsewhere, leading to illnesses so diverse in clinical type as to be unrecognizable except for the fact that they have conferred immunity to the disease, enable us to understand how impossible it was 17 years ago for the investigators of that Commission to identify among the natives a disease which could be the means of keeping the infection alive during the often long intervals between its appearances in epidemic form among the Syrians and Europeans.

E. H.

JAMES (S. P.). Les résultats des recherches récentes sur la fièvre jaune envisagés au point de vue des mesures destinées à empêcher la propagation de la maladie. [**The Results of Recent Work on Yellow Fever with Special Reference to Measures for preventing the Spread of the Disease.**]—*Bull. Office Internat. d' Hyg. Publique.* 1930. Dec. Vol. 22. No. 12. pp. 2278–2290. With 3 folding maps. [1 ref.]

A summary of recent work bearing on the methods of transmission and prevention with special reference to the problem of devising measures for preventing the spread of yellow fever. This problem is particularly important in view of the establishment of a number of air routes in both America and Africa, many of which pass through localities that have been known to be infected with yellow fever comparatively recently. Three maps show the routes and also indicate localities which have had cases of yellow fever within the last twenty years. The article concludes with a useful summary of all these localities given in tabular form.

E. H.

JAMES (S. P.). Quelques données récentes intéressant la prophylaxie de la fièvre jaune. [**Recent Observations on Yellow Fever Prophylaxis.**]—*Bull. Office Internat. d' Hyg. Publique.* 1930. Aug. Vol. 22. No. 8. pp. 1479–1480.

Attention is drawn to the results of BEEUWKES, BAUER and MAHAFFY (above, p. 283) on the prevalence of yellow fever in West Africa. With regard to the mosquitoes carrying the disease, it is found that the elimination of wells by providing town water supplies has been sufficient to cause the numbers of these insects to fall below the danger level. It is evident that yellow fever readily responds to prophylactic measures and is not among the more difficult diseases to combat.

E. H.

KLOTZ (Oskar) & BELT (T. H.). **The Identity of Yellow Fever Lesions in Africa and America.**—*Amer. Jl. Trop. Med.* 1930. Sept. Vol. 10. No. 5. pp. 299–304. [7 refs.] [Path. Dept., Univ., Toronto.]

The authors have compared the pathological changes in the tissues of 50 cases of yellow fever from West Africa with the tissues of 43 American cases. The so-called "Councilman necrosis" of the liver, to be described in another paper, was found to be constantly present in all the cases, as well as vacuolation and granular degeneration of the Kupffer cells. Cloudy swelling of the heart was another constant feature. Nuclear inclusion bodies were observed in 38 per cent. of the African cases, and only in 9·2 per cent. of the American, but the long formalin fixation in the case of most of the latter tissues may be a partial explanation of this discrepancy. The kidney is more severely affected in the African group although the lesions are of the same character in both groups. In general it is found that the nature of the pathological changes is identical in both African and American cases and any differences observed are compatible with the variations shown by different strains of the virus in both Africa and America.

E. H.

KLOTZ (Oskar) & BELT (T. H.). **The Pathology of the Spleen in Yellow Fever.**—*Amer. Jl. Path.* 1930. Nov. Vol. 6. No. 6. pp. 655–662. With 4 figs. on 2 plates. [4 refs.]

—— & ——. **The Pathology of the Liver in Yellow Fever.**—*Ibid.* pp. 663–688. With 1 chart in text & 1 coloured plate. [2 pages of refs.]

—— & ——. **Regeneration of Liver and Kidney following Yellow Fever.**—*Ibid.* pp. 689–697. [36 refs.] [Dept. of Path. & Bact., Univ., Toronto.]

These three articles are all concerned with the pathological changes in yellow fever, and add to the voluminous literature on the subject. The first two papers comprise a more extensive study of the histopathology of the spleen and liver, and the results agree with previous descriptions by KLOTZ and SIMPSON [this *Bulletin*, Vol. 25, p. 112]. It is of interest that the spleen shows none of the lesions characterizing the liver pathology of the disease. The cytoplasmic necrosis is lacking, fatty degeneration is negligible and there are no specific intranuclear inclusion bodies. In the yellow fever of Africa and America the most characteristic liver lesion is the acidophile degeneration in the cytoplasm of the parenchymal cells. This so-called Councilman lesion is very constant and is especially valuable in the diagnosis of human cases, which often do not show the intranuclear bodies. It cannot be confused with any other type of liver necrosis and the authors consider that in early stages it bears a certain similarity to the cytoplasm inclusions of other virus diseases. The mid-zonal distribution of these lesions in the lobules cannot be explained although the lesions themselves seem due to some toxic agent in the blood. Specific nuclear inclusions were found in the livers of 17 out of 19 *M. rhesus* infected with yellow fever, and in 23 out of 93 human cases of the disease.

The difficulty of distinguishing Weil's disease and yellow fever by clinical symptoms has led to a certain amount of confusion in previous descriptions of the pathology of the disease and the records of interstitial haemorrhage are probably a result of this confusion. Similarly wandering-cell infiltration has probably been described from cases of Weil's disease mistaken for yellow fever, as the authors found that exudative reactions were absent in all uncomplicated cases of the latter.

Referring to KUCZYNSKI and HOHENADEL'S observations [this *Bulletin*, Vol. 26, p. 296], in which they claimed to have identified a bacillus as the causal agent of yellow fever, the present authors state that the experimental lesions they described in their infected animals are not characteristic of this disease.

The third article contains particulars of the regeneration of the liver and kidney in 6 rhesus monkeys, 16 to 66 days after they had recovered from an attack of yellow fever. In every case complete and scarless regeneration occurred, which supports the clinical evidence that neither cirrhosis of the liver nor contracted kidney follows yellow fever in man. Except in chloroform poisoning there is no condition producing liver destruction of equal magnitude in which scar formation is not observed. The absence of fibrosis is said to be due to the peculiar immunity of the stroma structures to yellow fever ; there is no stimulation of connective tissue cells during the acute stage of the disease. The reasons for this may be the non-inflammatory, non-autolytic character of the pathological condition and to the absence of thrombosis in the small parencyhmal vessels. Regeneration originates in islands

of parenchymal cells which have survived the attack, and quickly restore the tissues to their original state.

E. H.

BURKE (A. W.) & DAVIS (N. C.). **Notes on Laboratory Infections with Yellow Fever.**—*Amer. Jl. Trop. Med.* 1930. Nov. Vol. 10. No. 6. pp. 419–426. [2 refs.] [Yellow Fever Lab., Internat. Health Division, Rockefeller Foundation, Bahia, Brazil.]

An account of three certain laboratory infections with yellow fever and also a probable one among Americans in Bahia, Brazil. The first of these patients was accidentally bitten by mosquitoes carrying the West African Asibi virus. He had received three 2 cc. doses of immune serum, the last about 4 months before this infection. The patient had a mild attack and showed no signs of albumin or casts in the urine and no jaundice. Two monkeys inoculated with blood taken 18 hours after the onset of illness had fever on the 5th day, but neither first nor second passage animals died. However, mosquitoes fed both on the patient and on one of these passage monkeys produced a fatal infection.

The second patient had received 2 cc. of a liver vaccine six months previously. The attack started suddenly and on the first day of fever he was given 14 cc. of human convalescent serum intravenously and the following day 18 cc. of immune serum from monkeys subcutaneously. The third day of the disease 20 cc. of yellow fever monkey immune serum were given in the early morning and later 50 cc. of anti-*icteroides* serum. The patient died on the evening of the fourth day of the disease. It is not known how this infection was acquired but the patient had performed an autopsy on an infected monkey 12 days before the onset of fever and also had bled an infected monkey 2 days before.

The third patient had lived in South and Central America for six years and had received 6 cc. of human convalescent serum 40 days before the onset of illness. He had a mild but typical attack of the disease which persisted for 4 to 5 days. Injections of human convalescent serum were given during the attack. It is not certain how the infection was acquired, but 4 days before the onset he had helped in drying some infected blood, and also about four weeks preceding the illness had lived in a hotel in the centre of the old endemic yellow fever district. No known cases had occurred in this neighbourhood for eight months but nevertheless the infection may have been present.

The fourth case received 16 cc. of human yellow fever immune serum only 23 days before the onset of illness. He had a very mild attack of yellow fever and albumin never appeared in the urine in sufficient amount to give a reliable quantitative test. During convalescence there was a faint trace of icterus. This patient had bled infected monkeys before the illness and had done serological work with the samples, but as in the two preceding cases the exact method of infection is not clear. In every case the nature of the disease was proved by inoculations into monkeys. The patient who died was the oldest of the four (50 years of age), had received no prophylactic human serum, and had been the shortest time in the tropics. The clinical symptoms, including the appearance of albumin and casts in the urine within less than 24 hours of the onset of fever, suggest that he may have had some previous renal involvement, but there is no definite evidence to this effect. Two patients (presumably the authors themselves) who received

immune serum after the onset of disease felt that it was of undoubted therapeutic benefit, as the fever subsided after its administration.

E. H.

LOW (G. Carmichael) & FAIRLEY (N. Hamilton). **Observations on Laboratory and Hospital Infections with Yellow Fever in England.**—*Brit. Med. Jl.* 1931. Jan. 24. pp. 125–128. With 1 text fig. [5 refs.]

The grave risk attaching to the routine collection and examination of human blood in yellow fever is strikingly illustrated by this important article, which contains full details of three laboratory infections briefly referred to in a previous publication [HINDLE, *ante* p. 285]. The first patient presumably became infected through handling yellow fever monkeys, but the two others never came in contact either directly or indirectly with infected animals; they acquired the disease one in making blood slides and performing a white cell count, and the other in doing routine biochemistry on a specimen of blood sent to the laboratory. All three patients showed typical infections and one of them died five days after the onset of fever. In this case there was no evidence that the administration of convalescent serum on the first day of illness nor the injection of calcium lactate had any effect on the course of the disease. The blood of the other two patients was inoculated into monkeys respectively 64 and 89¼ hours after the onset of the disease, and in both instances fatal infections were produced, although in the case of the latter patient 30 cc. of human convalescent serum had been administered the previous day.

It is evident therefore that there is considerable danger in the routine examination of blood in yellow fever cases during the first four days of illness, and in addition almost certainly during the incubation period. Consequently the authors recommend that in endemic areas, when the disease is prevalent, gloves should invariably be worn when taking blood, whether yellow fever is suspected or not.

E. H.

DINGER (J. E.), SCHÜFFNER (W. A. P.), SNIJDERS (E. P.) & SWELLENGREBEL (N. H.). Onderzoek over gele koorts in Nederland (vijfde mededeeling). [**Research on Yellow Fever in Holland.**]—*Nederl. Tijdschr. v. Geneesk.* 1930. Oct. 25. 74th Year. 2nd Half. No. 43. pp. 5253–5266. With 26 figs. on 1 folding plate. [17 refs.]

The 1st, 2nd, 3rd and 4th communications have already been noticed (this *Bulletin*, 1929, p. 1006; 1930, pp. 485, 486 & 869). This fifth communication deals with the pathological anatomy of the disease in monkeys. Yellow fever is sometimes difficult to diagnose in man and is especially liable to be confused with leptospirosis icterohaemorrhagica. It is still more difficult in monkeys. In this research post-mortem examinations were conducted on 76 rhesus monkeys, dead of yellow fever, and on 20 control animals of which one was normal and the remaining 19 had died of other diseases. In addition 16 cynomologus monkeys and one nemestrinus with 3 controls were examined post-mortem. The material used, therefore, was on a considerable scale. The course of infection, in general, was most rapid with blood injection, less so with liver injection and still less so by mosquito bite. Of the

changes produced by the disease in the test animals the most significant were jaundice, skin haemorrhages, lung haemorrhages, blood in the stomach with mucosal haemorrhage and haemorrhagic erosion, a typical liver, congested spleen and granular pale kidney. The most characteristic of all the macroscopic changes are in the liver, which was yellow to yellowish-brown, friable, and when cut left a greasy smear upon the knife. Microscopically also the liver changes are the most important for diagnosis. In frozen sections of the liver a diffuse fatty degeneration was evident, which however was less intense around the central vein to a depth of 1 or 2 cells and thus gave rise to a wreath-like appearance. Necrosis in these monkeys was comparatively slight. Of the control animals none showed any appearance that could not easily be differentiated from that of the test animals except two dead of phosphorus poisoning. Paraffin sections of the liver in the yellow fever animals showed the general architecture of the liver to be not greatly altered. In the lobular columns there were more or less acidophile necrotic cells among the fatty cells. A very characteristic appearance was the acidophile intranuclear degeneration forming the inclusion bodies of TORRES, which may be looked on as diagnostic. These bodies, finely granular, butterfly shaped or half-moon shaped, have no connexion with the nucleolus and are found in greater numbers the greater the necrosis present. That they are indicative of the presence of the virus in the nucleus seems very doubtful. Their numbers in the liver were not proportional to the severity of the infection and they showed no evidence by any method of any more specifically staining central body. No leptospiras were found by Levaditi's method. Illustrations which accompany the article show skin petechiae, gastric haemorrhage and haemorrhagic erosion, fatty degeneration of the liver and the cellular sheath to the central vein, acidophile necroses and accompanying polynuclear infiltration and the specific Torres bodies.

W. F. Harvey.

DINGER (J. E.), SCHÜFFNER (W. A. P.) & SNIJDERS (E. P.). Untersuchungen ueber Gelbfieber in den Niederlanden. Impfversuche an Meerschweinchen. [**Dutch Studies on Yellow Fever. Inoculation Experiments with Guineapigs.**]—*Zent. f. Bakt.* I. Abt. Orig. 1930. Dec. 8. Vol. 119. No. 1/2. pp. 1–11. [4 refs.] [Colonial Inst., Amsterdam.]

The authors have made a careful series of experiments, graphically expressed in tabular form, on the susceptibility of the guineapig to yellow fever. Their results, to some extent, agree with those of KUCZYNSKI and HOHENADEL [see this *Bulletin*, Vol. 27, p. 478], who were able to obtain a modified form of the disease in guineapigs by the inoculation of infected material from monkeys. The present authors find that a disease can be produced in guineapigs and carried on through a series of animals, but after the second guineapig passage the virus became modified to such an extent that it was no longer possible to infect monkeys from the guineapigs. Thirty-seven guineapigs were inoculated with material from infected monkeys and of these 20 died, 9 with haemorrhages in the stomach, 3 with peritonitis, 4 with both these conditions, 1 with pseudotuberculosis, and 3 with no obvious pathological changes. The disease could also be transmitted from infected to normal guineapigs. Of 10 inoculated with infected liver material, 5 died, three with haemorrhages in lungs and stomach, and

two with peritonitis. Thirty-eight were inoculated with infected blood, of which 18 died, 9 with haemorrhages in lungs and stomach, 8 with peritonitis and one with no obvious pathological conditions; 2 were inoculated with peritoneal fluid and both died of peritonitis. Two guineapigs were infected by the bites of mosquitoes that had fed on a yellow fever monkey. They both died after 19 and 20 days, with stomach and lung haemorrhages. Mosquitoes that had fed on infected guineapigs were also found capable of transmitting the infection. All attempts to infect monkeys from guineapigs after the first passage gave negative results, and also all attempts to infect monkeys by the bite of mosquitoes that had fed on infected guineapigs.

E. H.

DAVIS (Nelson C.). **The Susceptibility of Marmosets to Yellow Fever Virus.**—*Jl. Experim. Med.* 1930. Sept. 1. Vol. 52. No. 3. pp. 405–415. With 1 plate. [5 refs.] [Yellow Fever Lab., Internat. Health Division, Rockefeller Foundation, Bahia, Brazil.]

The author has infected with yellow fever two species of marmosets, *Callithrix albicollis* and *Leontocebus ursulus*, by the bites of infected mosquitoes and also by the inoculation of infected blood. Moreover, the virus was passed back into rhesus monkeys after a varying number of passages through these marmosets. There is some indication that after a series of passages through *Callithrix albicollis* the virus loses some of its virulence for *Macacus rhesus*. Very few of these marmosets showed a definite fever and in the case of *albicollis* the lesions found at autopsy did not resemble those of yellow fever as seen in man, in *Macacus rhesus*, and in other species of monkeys. The convalescent serum of five that recovered was found to protect rhesus monkeys against yellow fever whilst the normal serum had no effect. In the case of the tamarins, *Leontocebus ursulus*, five were infected, of which four developed fever and all died. At least two showed liver changes comparable with those found in human beings and rhesus monkeys dead of yellow fever. It is conceivable that these little monkeys, which are often kept as pets, may have some importance in the spread of the disease in nature.

[KUCZYNSKI and HOHENADEL were the first to demonstrate the susceptibility of marmosets to yellow fever (see this *Bulletin*, Vol. 27, p. 87).]

E. H.

PHILIP (Cornelius B.). **Possibility of Mechanical Transmission by Insects in Experimental Yellow Fever.**—*Ann. Trop. Med. & Parasit.* 1930. Dec. 18. Vol. 24. No. 4. pp. 493–501. [7 refs.]

In view of the high concentration of yellow fever virus in the blood of infected monkeys, it might have been expected that mechanical transmission by biting insects would be liable to occur. The author has made careful experiments to test this point, using *Aedes aegypti* and *Cimex lectularius*. The insects were fed partially on an infected monkey and allowed to complete their meal, without any appreciable interval, on normal monkeys. Negative results were obtained in three experiments in which 39 to 100 mosquitoes were used. Nine adult bed-bugs and 50 larval and nymphal stages also failed to infect a normal rhesus monkey, during seven alternate transfers between the normal monkey

and an infected one during one feeding. Although 100 monkey lice (*Pedicinus* sp.), when removed from an infected monkey and immediately ground up and inoculated into a normal one, produced infection, there is no evidence that these lice are ever capable of transmitting the disease. The results of these experiments clearly indicate that there is no likelihood of yellow fever being spread mechanically by the bites of insects.

E. H.

FINDLAY (G. M.) & HINDLE (E.). **Guanidine-like Substances in the Blood in Experimental Yellow Fever.**—*Lancet.* 1930. Sept. 27. pp. 678–679. [13 refs.] [Wellcome Bureau of Scientific Research, London.]

The symptoms of yellow fever seem to be due rather to the toxins resulting from the lesions originated by the virus than to the virus itself, and with this object the authors have studied the toxins in order to determine methods for their neutralization. One of these toxins is shown to be a guanidine-like substance, the quantity of which increases in the blood of monkeys infected with yellow fever from an average of about 0·2 mgm. per 100 cc., up to more than double this amount. The toxic symptoms characteristic of this disease closely resemble those recorded as a result of guanidine poisoning, and also of carbon tetrachloride poisoning. Moreover, diminution in the blood sugar also occurs in these conditions as well as in yellow fever.

Calcium salts are known to have a highly protective action in treating intoxications associated with an increase in the guanidine base content of the blood, at the same time raising the blood sugar.

Consequently injections of 5 cc. of a 5 per cent. solution of calcium lactate were given to a monkey infected with yellow fever and after two hours the guanidine base content was found to have been reduced from 0·400 to 0·251 mgm. per 100 cc. of blood. Five hours later the quantity had again risen to 0·498 mgm., so it is evidently necessary to give injections at intervals of four to six hours. When this was done the guanidine content was kept down, but unfortunately, even when combined with glucose, it did not prevent the final collapse and death in monkeys suffering from yellow fever. These monkeys, eight in number, all showed typical necrosis of the liver, but in contrast with untreated animals, none of them showed any gastro-intestinal or pulmonary haemorrhages. Consequently there is some evidence that repeated injections of calcium may be of some use in preventing haemorrhage in yellow fever cases. Finally, the authors suggest that in view of the extremely low blood pressure, possibly associated with the presence of toxic amines in the blood, the intravenous injection of saline or of gum arabic, as used in cases of surgical shock, might be of value in the treatment of human cases of yellow fever.

E. H.

WAKEMAN (A. Maurice) & MORRELL (Clare A.). **Chemistry and Metabolism in Experimental Yellow Fever in *Macacus rhesus* Monkeys. II. Nitrogen Metabolism.**—*Arch. Intern. Med.* 1930. Sept. Vol. 46. No. 3. pp. 382–401. With 2 charts. [12 refs.]

The authors continue their observations on the study of changes in the blood and urine of yellow fever monkeys [see this *Bulletin*, Vol. 27, p. 876]. Their results indicate that the non-protein nitrogen, amino-acid, urea and

" rest " nitrogen of the blood increase considerably towards the end of the infection. The blood creatinine, uric acid and ammonia show little, if any, change. The amino acids increase both in absolute amount and in proportion to non-protein nitrogen, while the urea decreases in proportion to the latter. A few cases showed an absolute decrease in blood urea nitrogen. These changes were only found towards the end of fatal infections and did not occur during the early stages of the disease nor in those monkeys which recovered.

There were no marked changes in the partition of these substances in the urine. The changes during the terminal period are much more pronounced when the accumulation of these substances in the body fluids is taken into consideration. Organic acid and phosphorus excretion usually increase considerably during the disease.

All the metabolic changes observed can be interpreted as resulting from a loss of liver function and there was no definite evidence of serious impairment of kidney function, except a terminal anuria probably due to extreme reduction of blood pressure.

E. H.

SMITH (E. C.). **Hepatic Intranuclear Changes in Yellow Fever.**—*West African Med. Jl.* Lagos. 1930. Oct. Vol. 4. No. 2. pp. 45–46. With 4 figs (1 coloured) on 2 plates. [2 refs.] [Med. Research Inst., Lagos.]

The author found the characteristic acidophile intranuclear inclusions in the liver cells of 39 out of 40 *Macacus rhesus* infected with yellow fever, whilst in 10 normal monkeys these bodies were never found. The livers of 22 West African human cases were also examined, and of these 15 showed well-marked inclusions, whereas in 8 cases of death from other causes, blackwater fever, obstructive jaundice, relapsing fever, etc., no such bodies were found. [It is of interest that West African cases of the disease seem to show these inclusion bodies much oftener than Brazilian ones.]

E. H.

TORRES (C. Magarinos). Dégénérescence oxychromatique et fièvre jaune chez le singe. [**Oxychromatic Degeneration and Yellow Fever in the Monkey.**]—*C.R. Soc. Biol.* 1930. Dec. 12. Vol. 105. No. 34. pp. 727–729. With 4 text figs. [1 ref.]

——. Dégénérescence oxychromatique et la fièvre jaune chez l'homme. [**Oxychromatic Degeneration and Yellow Fever in Man.**]—*Ibid.* pp. 732–733. With 2 text figs. [1 ref.]

i. The first paper comprises further details on the nature of the intranuclear bodies developed in the liver cells of monkeys infected with yellow fever. The author disagrees with COWDRY and KITCHEN's account of these bodies [see this *Bulletin*, Vol. 27, p. 475]. He finds that the inclusions never present the aspect of individual acidophile granules, but consist of a " fundamental substance " and a stroma. The first is formed by oxychromatin and various nucleoproteins and stains pink with eosin, phloxin, and carmin ; it does not stain with either methyl green or methylene blue. The fundamental substance shows unequal colouration owing to the stroma, which seems derived from the inner network of the nucleus.

ii. The second paper is concerned with these bodies in man, but of 17 yellow fever cases examined intranuclear bodies were found only in the liver cells of 3 patients : a four-year-old girl who died 40 hours after the first appearance of symptoms ; a soldier who died on the second day of the disease ; and a young man (19 years) who died on the sixth day. The inclusions resemble those present in yellow fever monkeys, but it is difficult to recognize early stages, owing to nonspecific alterations in the nucleoplasm causing the appearance of acidophile granulations which may be confused with those due to yellow fever.

E. H.

STEFANOPOULO (G. J.) & CODOUNIS (A.). Sur la protéinémie du singe et particulièrement du macaque atteint de fièvre jaune expérimentale. [**On the Proteinaemia of the Monkey and in particular of Macacus infected with Yellow Fever.**]—*C.R. Soc. Biol.* 1930. Nov. 4. Vol. 105. No. 29. pp. 269–271. [3 refs.]

The authors have studied the protein content of blood of normal monkeys, and also others inoculated with yellow fever, some after having been vaccinated. Out of seven large baboons, three normal individuals gave figures ranging from 65·52 to 68·49 for the total protein content of the blood, whilst four immunized baboons (3 against poliomyelitis, and one against yellow fever) gave figures ranging from 63·77 to 94·56.

Nine *Macacus rhesus* were also examined. The results of the examination of five of them before and after various infections are given in a table. It is of especial interest that although infection with yellow fever caused a marked decrease in the total proteins, vaccination with a formolized yellow fever vaccine produced no change in the protein content. These monkeys were subsequently inoculated with yellow fever virus and found to be protected. On the other hand the baboons that had been inoculated repeatedly with yellow fever virus and developed antisera, showed an increase in the protein content.

E. H.

DINGER (J. E.), SCHÜFFNER & SNIJDERS. Untersuchungen über den Erreger des Gelbfiebers. Züchtungsversuche. [**Studies on the Aetiology of Yellow Fever. Culture Experiments.**]—*Zent. f. Bakt.* I. Abt. Orig. 1931. Jan. 20. Vol. 119. No. 5/6. pp. 304–311. [9 refs.]

——, —— & ——. Onderzoek naar den verwekker der gele koorts. —*Nederl. Tijdschr. v. Hyg., Microbiol. en Serol.* 1931. Vol. 5. No. 3. pp. 160–171. [11 refs.]

The authors have made a very exhaustive series of experiments on the cultivation of yellow fever material, mainly with the object of deciding the aetiological significance of *Bacillus hepatodystrophicans*, described by KUCZYNSKI and HOHENADEL [this *Bulletin*, Vol. 27, pp. 478 & 482] as the causative organism of this disease. Although various bacteria were occasionally cultured from yellow fever tissues, it is shown that these are generally diphtheroids derived from the air, and tubes containing yellow fever tissues in Kuczynski's medium, when infected experimentally by breathing in them or exposure to air, showed a growth of a diphtheroid bacillus which could not be distinguished from *B. hepatodystrophicans*. Monkeys inoculated with these cultures and also with cultures received from Professor KUCZYNSKI showed no signs of any infection with yellow fever and no immunity. Yellow fever virus was found to live in culture tubes containing this bacillus for 2 days at 37° C. The authors conclude that this diphtheroid bacillus is identical with *B. hepatodystrophicans* and is derived from the air ; it has no significance therefore in the aetiology of yellow fever.

E. H.

BUCHBINDER (Leon). **Yellow Fever : a Filtrable Virus Disease.**—*Arch. Pathology.* 1930. Oct. Vol. 10. No. 4. pp. 589–603. [92 refs.] [College of Physicians & Surgeons, Columbia Univ., New York.]

A comprehensive and accurate summary of recent work on yellow fever, giving an excellent idea of the present position of our knowledge of the subject.

E. H.

BARROSO (Sebastião). Quelques aspects de la campagne sanitaire contre la fièvre jaune à Rio de Janeiro. [**Certain Aspects of the Sanitary Campaign against Yellow Fever at Rio de Janeiro.**]—*Rev. Sud-Américaine de Méd. et de Chirurg.* Paris. 1930. Oct. Vol. 1. No. 10. pp. 1015–1019.

After drawing attention to the great financial losses involved in past yellow fever epidemics, the author discusses the sanitary methods that have been used against them and clearly shows the superior efficacy of those used in the recent epidemic.

E. H.

ARAGAO (Henrique de Beaurepaire). Possibilité de la transmission directe de la fièvre jaune, de Stégomyie à Stégomyie.—*C.R. Soc. Biol.* 1929. Nov. 8. Vol. 102. No. 29. pp. 474–475.

ARAGAO (Henrique de Beaurepaire). Infection des *Aedes aegypti* males avec les virus de la fièvre jaune.—*C.R. Soc. Biol.* 1929. Nov. 8. Vol. 102. No. 29. p. 476. [1 ref.]

ARAGAO (H. de Beaurepaire) & LIMA (A. da Costa). Transmission de la fièvre jaune par l'application de fèces de moustiques infectés sur la peau et la muqueuse oculaire intactes.—*C.R. Soc. Biol.* 1929. Nov. 8. Vol. 102. No. 29. pp. 477–478. [1 ref.]

ARAGAO (H. de Beaurepaire) & LIMA (A. da Costa). Sur le temps minimum nécessaire aux Stégomyies infectées pour excréter des fèces virulentes. —*C.R. Soc. Biol.* 1929. Nov. 8. Vol. 102. No. 29. pp. 478–480.

BARROSO (Sebastião). A febre amarella no Brasil. A nova phase da campanha. —*Brasil-Medico.* 1930. Mar. 29. Vol. 44. No. 13. pp. 374–376.

DUREN (A. N.). Les mesures prises contre la fièvre jaune à Matadi en 1928.—*Bull. Office Internat. d'Hyg. Publique.* 1930. Feb. Vol. 22. No. 2. pp. 287–306. With 2 charts in text. [1 ref.]

HOFFMANN (W. H.). Die Ätiologie des Gelben Fiebers.—*Seuchenbekämpfung.* 1929. Vol. 6. No. 4. pp. 234–237.

HOFFMANN (W. H.). Die Immunitätsverhältnisse beim Gelbfieber und ihr Einfluss auf die Epidemiologie.—Reprinted from *Immunität. Allergie und Infektionskr.* 1929. Vol. 1. No. 12. pp. 377–386. [Finlay Inst., Havana, Cuba.]

JORGE (Ricardo). A febre amarella e a campanha sanitaria no Rio de Janeiro (1928–1929).—*Brasil-Medico.* 1930. June 14 & 21. Vol. 44. Nos. 24 & 25. pp. 649–653; 676–680.

LEGER (Marcel). La transmission de la fièvre jaune d'après les données récentes. —*Gaz. hebd. Sci. Méd. de Bordeaux.* 1930. Feb. 2. Vol. 51. No. 5. pp. 65–70. [18 refs.]

LE PRINCE (H.). Sur les mesures qui peuvent empêcher la propagation de la fièvre jaune.—*Bull. Office Internat. d'Hyg. Publique.* 1930. Dec. Vol. 22. No. 12. pp. 2291–2294.

LINS (Sinval A.). A febre amarella através um seculo de observação clinica e orientação scientifica.—*Brasil-Medico.* 1930. Mar. 1 & 8. Vol. 44. Nos. 9 & 10. pp. 280–288. [41 refs.]

PICHAT (Jean). Note sur l'urine des sujets atteints de fièvre jaune.—*Marseille-Méd.* 1929. Sept. 25. Vol. 66. No. 27. pp. 372–374.

RELAPSING FEVER AND OTHER SPIROCHAETOSES.

CAZANOVE. La fièvre récurrente à poux dans les colonies françaises de l'Afrique occidentale et centrale.—*Bull. Office Internat. d'Hyg. Publique.* 1930. Nov. Vol. 22. No. 11. pp. 2135–2142. [1 ref.]

LASNET. La fièvre récurrente à poux en Afrique Française. [**Louse transmitted Relapsing Fever in the French African Colonies.**]—*Ann. de Méd. et de Pharm. Colon.* 1930. July–Aug.–Sept. Vol. 119. No. 3. pp. 408–417.

These two papers contain an account of the incidence of relapsing fever in the French African colonies from 1924 to 1928 inclusive, in continuation of GOUZIEN's report to the end of 1923.

In French Equatorial Africa, in the region of Chad, the situation seems a little uncertain and it is difficult to say whether the disease will disappear as in Nigeria, or persist in isolated regions as in the Upper Volta. In French West Africa this persistent centre of infection in the Upper Volta is of great importance from a prophylactic point of view, as it may constitute a source of future epidemics. In addition the disease has become established in Senegal, in the Cayor region near the sea, and in spite of free communication, it does not seem to spread inland. The persistence of the infection in this neighbourhood is attributed to the presence of small wild rodents, which serve as a reservoir for the virus. [In the absence of Ornithodorus, which have not been found in this part of Africa, the manner in which rodents would serve as a reservoir for a louse transmitted relapsing fever is not quite evident.]

E. Hindle.

LASNET. Communication sur la marche de la fièvre récurrente en Afrique centrale. [**The Spread of Relapsing Fever in Central Africa.**]—*Bull. Acad. Méd.* 1930. July 22. Year 94. 3rd Ser. Vol. 104. No. 29. pp. 112–114.

This disease broke out in " the Upper Guinea " about ten years ago, probably introduced by natives from the Mediterranean region ; it then spread across to the Sudan [see this *Bulletin*, Vol. 27, p. 691]. The first cases were observed in 1921 at Kouroussa among Moroccan and Algerian soldiers. It spread down the Niger and at Segou 1,200 deaths were recorded between December 1921 and March 1922. During this year it travelled east through the Dori region and is estimated to have caused 5,000 to 6,000 deaths. During 1923, small outbreaks occurred further west, but the main epidemic was towards the east, especially on each side of the road to Say. The mortality was not less than 14,000. The following year no cases were recorded in Guinea, but in the Upper Volta region and across to Koutiala the epidemic caused at least 20,000 deaths. In 1925, the outbreak died down in the Upper Volta and Sudan, but broke out in the region of Lake Chad and invaded the north of the Cameroons ; the mortality is estimated at 9,000 deaths. Subsequently the disease lasted until 1929 in the North of Equatorial Africa, but never spread to the South among the wild naked tribes of that region. About 10 per cent. of the population of the affected regions died of the disease, the mortality varying from 5 to 25 per cent. of the population. Although the epidemic has now ceased there are isolated endemic centres in the Upper Volta and Chad regions ; possibly the infection is maintained in animal reservoirs.

Novarsenobenzol has been generally used for treatment and has given very good results. Prophylaxis has been based on the destruction of body lice and the efforts of the sanitary authorities [combined with the general absence of clothing towards the south] have been successful in checking the further spread of this severe epidemic.

E. H.

DE PAOLI (Paolo). Osservazioni sopra un focolaio epidemico di febbre ricorrente in Eritrea. [**A Focal Epidemic of Relapsing Fever in Eritrea.**]—*Arch. Ital. Sci. Med. Colon.* 1930. May 1. Vol. 11. No. 5. pp. 303–309. English summary (5 lines) p. 310. [Regina Elena Colonial Hosp., Asmara.]

In July–October, 1929, several cases of relapsing fever were met with in Asmara. When looking for the possible source of infection the author noticed several persons, transient visitors and beggars, who appeared ill, and examination of their blood showed in a single day 6 of them to be harbouring spirochaetes. The exacerbations of fever lasted for 10 days at a time. It is believed that the disease had been introduced by travellers from Abyssinia. Search failed to discover any ticks, but *Pediculus vestimenti* was found on all the patients and *Cimex lectularius* where they slept.

H. H. S.

FRANCHINI (Giuseppe). Distribuzione degli Ixodidi nello Jemen (Arabia meridionale). Reperto di Spirochete nei pidocchi della città di Sana. [**Ticks in Jemen (Southern Arabia). Spirochaetes in Lice in Sana.**] —*Arch. Ital. Sci. Med. Colon.* 1930. Aug. 1. Vol. 11. No. 8. pp. 449–452. With 2 text figs. (1 map). English summary (6 lines).

Professor Franchini proceeding with his survey (see this *Bulletin*, Vol. 27, p. 925) has found four species of ticks in Jemen : *Amblyomma variegatum, Hyalomma aegyptium, Rhipicephalus evertsi* and *R. sanguineus*. All four were present in Hodeida on the coast, the second only in Taiz, this and the fourth in Sana (both inland towns). The same four species had been found in Eritrea. He examined by serial sections bugs, lice and ticks, and in the lice alone spirochaetes were present, and the author believes, therefore, that the relapsing fever of this district is louse-borne.

H. H. S.

KATZ (D. I.). Sur les espèces du typhus recurrens aux Pamires. [**Relapsing Fever in the Pamirs.**]—*Pensée Méd. d' Usbéquistane et de Turquménistane.* Tashkent. 1930. Aug.–Sept. No. 11–12. pp. 5–12. With 7 text figs. [In Russian.]

The author describes 38 cases of relapsing fever of the " Persian variety " observed in the Western part of the Pamir region bordering with India and Afghanistan. The elevation of the district is between 1,700 and 2,800 metres, but the disease does not occur above 2,000 metres. The condition was formerly diagnosed as a form of quinine-resistant malaria, but according to the author malaria is absent in this country. In typical cases the course of the disease is as follows : After an incubation period of 6–8 days there is a sudden rise of temperature to 39–40° C., which is maintained as long as the attack lasts. In untreated cases there are 7–9 attacks with apyretic periods of 6–24 days. The rise in temperature is accompanied by rigor, cyanosis, headache, pain in the lumbar region, and sometimes followed by vomiting.

The spleen and liver were not enlarged in all cases, but were sensitive to palpation. Scanty spirochaetes were present in all cases. The author found that neosalvarsan was most effective when administered by the "method of saturation," i.e., independently of the stage of disease, as follows: On the first day of attack or of apyrexy 0·3 gm., 3–4 days later 0·45 gm., 5–6 days later 0·6 gm., a total of three injections. As the result of treatment the number of attacks diminished considerably and they assumed a mild form. Irregular administration of neosalvarsan, on the other hand, only prolonged the apyretic period without any appreciable alleviation of the symptoms. C. A. Hoare.

WELLER (Burford) & GRAHAM (G. M.). **Relapsing Fever in Central Texas.**—*Jl. Amer. Med. Assoc.* 1930. Dec. 13. Vol. 95. No. 24. pp. 1834–1835.

This is the first definite record of the existence of tick-transmitted relapsing fever in the U.S.A., and is of especial interest since the authors were able to trace the source of infection. This was a cave in the Colorado River valley, about 75 miles above Austin, which contained very large numbers of *Ornithodorus turicata*. Three out of four boys who entered this cave and were bitten by these ticks developed typical relapsing fever exactly six days later and all suffered from three or more febrile attacks. One of the authors who also entered this cave was bitten by a tick and developed fever on the seventh day; spirochaetes were found in his blood 18 hours later and 1·3 gm. of neoarsphenamine was given intravenously. This had no obvious effect and a second dose of 0·6 gm. was given 18 hours later, which caused the temperature to fall at once and there were no relapses. This cave was frequented by goats and sheep and also by various wild animals, but had only very rarely been visited by human beings, so the infection must have been maintained in some animal host. Some of the ticks were fed on a rabbit which became ill on the sixth day and died with numerous spirochaetes in its peripheral blood. These are said to vary in length from 18 to 25 microns, with ten to fifteen spiral turns. E. H.

BANNISTER (Kimball). **Relapsing Fever** (*Febris recurrens*, **Ruckfallfieber, Spirillum Fever, Tick Fever**).—*Southwestern Med.* 1930. Dec. Vol. 14. No. 12. pp. 581–582.

The record of a case of relapsing fever which developed after the patient has spent a vacation in the neighbourhood of Greer, Arizona. The symptoms were typical and during the seventh febrile attack spirochaetes were found in the blood.

[Although the source of infection is not stated, the number of febrile attacks and the locality suggest the likelihood of ticks being responsible (see above).] E. H.

DELANOË (P.). Le rôle du porc-épic comme réservoir de virus du spirochète marocain, *S. hispanicum* var. *marocanum* Nicolle et Anderson, 1928. [**The Porcupine as a Reservoir of the Moroccan Spirochaete,** *S. hispanica* **var.** *marocana.*]—*C. R. Acad. Sci.* 1930. Dec. 29. Vol. 191. No. 26. pp. 1481–1484. [2 refs.]

The author examined 17 porcupines, 2 young and 15 adult, caught in Morocco, and by inoculating blood from each into a series of guineapigs was able in one instance to infect one of these animals with the spiro-

chaete of Moroccan relapsing fever. Seven adult porcupines were inoculated with large doses of this spirochaete, and all except one were completely immune; one individual showed a mild infection. These individuals showed a polyvalent immunity against the various immunologically distinct strains of spirochaetes occurring in Morocco, which suggests that the porcupines in wandering about are repeatedly infected by the Ornithodorus inhabiting their burrows.

E. H.

DELANOË (P.). Sensibilité du renard au spirochète marocain, *Sp. hispanicum* var. *marocanum* Ch. Nicolle et Ch. Anderson, 1928. Le renard réservoir de virus de ce spirochète. [**The Susceptibility of the Fox to Moroccan Relapsing Fever,** *S. hispanica* **var.** *marocana.* **The Fox as a Reservoir.**]—*C.R. Acad. Sci.* 1931. Jan. 12. Vol. 192. No. 2. pp. 116–119. [1 ref.]

In a previous article the author recorded the presence of infected Ornithodorus in the burrows of foxes. Two young Algerian foxes were inoculated intraperitoneally with blood containing the Moroccan spirochaete and both became infected, one showing a relapse. The infection was very mild and the blood ceased to be infective after 25 days and in one the nervous system failed to infect after 35 days. A third fox was infected by the bites of Ornithodorus and although spirochates could never be found, 2·5 cc. of its blood, collected on the tenth day, produced infection in two guineapigs. Three young Moroccan foxes were found naturally infected with this spirochaete as tested by blood inoculation into guineapigs. Two adult Moroccan foxes were found to be uninfected and the blood of one that was tested was found to be protective against the spirochaete, and also this individual was completely resistant to a large dose of infected blood.

The results indicate that in Morocco the young fox is susceptible to the local relapsing fever, and may serve as a reservoir of the disease, since it evidently becomes infected in nature.

E. H.

SOFIEFF (M. S.). Vergleichende serologische Untersuchungen an Stämmen der Recurrens-Spirochäten. [**Comparative Serological Researches on Strains of R.F. Spirochaetes.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1930. July. Vol. 34. No. 7. pp. 382–386. [Inst. for Ship & Trop. Diseases, Hamburg.]

SAGEL (W.). Ueber die wechselseitigen Beziehungen verschiedener Recurrensstämme, besonders von *Spirochaeta Duttoni* und *Spirochaeta Berbera* (Marokko) nach neueren Erfahrungen an Paralytikern. (Ein Nachtrag zur Arbeit von Sofieff in Heft 7 dieser Zeitschrift.)—*Ibid.* Aug. No. 8. pp. 429–439.

The first author has compared four strains of relapsing fever spirochaetes, viz.:—*S. duttoni*, Hamburg, obtained by FÜLLEBORN and MAYER in East Africa in 1906, and maintained in ticks and white mice; *S. hispanica*, obtained from infected *Ornithodorus marocanus* received from Spain in 1927; a Moroccan strain (*S. berbera* ?), isolated from a patient in 1927 and maintained in mice; and finally an Angola strain, isolated from a patient in 1928 and subsequently kept in mice. Adhesion, agglutination and cross immunity tests were made with these strains. The results show that the Moroccan strain and the Hamburg

strain of *S. duttoni* were indistinguishable, but were different from the Angola and *hispanica* strains. The two latter were also distinct from each other. The strain of *S. duttoni* and the Moroccan strain were used by SAGEL in the treatment of general paralysis [see this *Bulletin*, Vol. 25, p. 591] and in man were found to be distinct. In mice, however, the two strains have become indistinguishable, a fact which is very difficult to explain.

ii. The second article contains full details of the changes that have occurred in these two strains during the past two years, based on inoculations into human subjects, and also further particulars about the other two. All four strains have retained their pathogenicity towards man. The *duttoni* and Moroccan strains produced persistent brain infections in the mouse, whilst the other two did not. After passages through the brain of the mouse the two former strains seem to have become indistinguishable.

When patients that had recovered from infection with a strain of *S. duttoni* were subsequently inoculated with the original Moroccan strain (*S. berbera*) they all showed spirochaetes in the blood, but after this strain had passed through the brain of a mouse, it no longer produced any infection when inoculated into patients immune against *S. duttoni*. The *hispanica* strain tested in these immune patients four times failed to produce infection and five times succeeded. The Angola strain produced infection in all the patients tested who were immune against the other strains. Tables are given illustrating the results of various immunity tests in human patients. Four patients immune against the original Moroccan strain were subsequently inoculated with *S. duttoni* after passage through a mouse brain. Two of them became infected and two were resistant. Later 5 patients immunized against a brain passage strain of *S. berbera* were all resistant against a similar strain of *S. duttoni*, but were susceptible to the *hispanica* and Angola strains. But of 7 patients immunized against the *hispanica* strain, three became infected and two were resistant when subsequently inoculated with a brain passage strain of *S. berbera*, also an inoculation of the original *berbera* strain produced infection. All these patients were resistant to a subsequent inoculation of *S. duttoni* after brain passage, whilst one individual who had only received an injection of *hispanica* showed a very heavy infection when inoculated with this *duttoni* strain. Patients immunized against the Angola strain were half of them resistant to a brain passage strain of *S. berbera*, whilst the other half became infected; those tested were all susceptible to *hispanica*.

The results show that marked changes are undergone by these organisms as a result of brain passage in the mouse. In addition to the disappearance of any serological distinction between the two strains, they seem to have increased in virulence to man as a result of the brain passage. A similar increase in virulence has also been observed in the *hispanica* strain as a result of passage in human patients. E. H.

PLAUT (F.) & GRABOW (C.). Ueber die Rolle der Schutzstoffe des Blutes für die kritische Beendigung der Fieberanfalle und für die Entwicklung der Immunität bei der Impfrekurrens des Menschen. [**Inoculated Relapsing Fever: Rôle of Protective Substances in the Blood for the Termination of Febrile Attacks, and in the Development of Immunity.**]—*Ztschr. f. Immunitätsf. u. Experim. Therap.* 1930. Vol. 68. No. 5/6. pp. 494–509. With 6 charts in text. [11 refs.]

The authors have studied the development of immune bodies in the blood of six patients infected with a strain of *S. duttoni* and obtained

very interesting results suggesting that the termination of each febrile attack is not dependent on the production of antibodies. The serum of these patients was collected at various intervals and protection tests made in mice. The method of applying these tests was to inactivate the serum at 56° C., then mix various dilutions with infected mouse blood so that each microscopic field contained one spirochaete, and finally 0·1 cc. of guineapig serum was added for complement. The mixture was left at room temperature for 15 minutes and then inoculated intraperitoneally into white mice.

The results indicate that after the earlier relapses the serum had no protective action and in one individual there was none even after four febrile attacks. Then the serum gradually acquired a protective action which increased after successive relapses until after intervals varying from 23 to 60 days it became strongly protective. This high concentration of immune bodies very soon diminished and generally was found to disappear within one or two years, sometimes earlier. Although the blood of these patients may be free from antibodies, they are refractory against reinfection. These facts show that the disappearance of spirochaetes is not dependent on the protective substances in the blood, and their sudden death at the end of each attack is not yet explained.

E. H.

LEVADITI (C.), ANDERSON (T.), SELBIE (F. R.) & SCHOEN (R.). L'encéphalite récurrentielle. [**Encephalitis of Relapsing Fever.**]—*Bull. Acad. Méd.* 1930. June 17. Year 94. Ser. 3. Vol. 103. No. 24. pp. 672–690. With 12 text figs. [5 refs.]

It has been shown by the authors that relapsing fever spirochaetes [a Brazzaville strain of *S. duttoni* was used] will persist in the central nervous system of immune animals such as mice, rats, monkeys and rabbits. Sooner or later the majority of such animals show a type of encephalitis resembling that of general paralysis. A detailed description is given of the pathological changes in the brains of rabbits and various species of monkeys, which had been exposed to infection with *S. duttoni* and subsequently showed residual brain infections and signs of encephalitis. Occasionally the encephalitis was present although the brain had ceased to be infective, but in the majority of cases the two occurred together. In spite of prolonged examination with dark ground illumination, and of silver stained tissues, the authors never succeeded in finding spiral forms in the brains of rabbits and monkeys showing marked encephalitis, yet these tissues were proved to contain the spirochaetal infection by inoculation experiments in mice.

By a study of rats infected with *S. duttoni* the authors have traced the rare invasion of the brain parenchyma by individual spirochaetes, which disappear at the same time as those in the blood; although spirochaetes cannot be seen in such brains, yet they remain virulent for some months in some invisible form. This invisible stage of relapsing fever spirochaetes cannot be preserved in glycerine, and also cannot be separated from the nervous tissue by filtration.

E. H.

MATHIS (C.) & DURIEUX (C.). Persistance de *Sp. Duttoni*, var. *crocidurae* dans le cerveau et dans la rate de la souris infectée expérimentalement. [**The Persistence of** *S. duttoni* **var.** *crocidurae* **in the Brain and Spleen of Infected Mice.**]—*Bull. Soc. Path. Exot.* 1930. Oct. 8. Vol. 23. No. 8. pp. 862–866. [Pasteur Inst., Dakar.]

The series of experiments performed by the authors show that this strain of *S. duttoni*, originally isolated from a shrew mouse at Dakar, may persist in the brains of infected mice up to 235 days after being inoculated. Brain infection seems to be a constant feature of this spirochaete and always persists for several weeks; consequently the number of animal passages, necessary for keeping the virus, can be much reduced. Out of 46 inoculated mice only one failed to show a brain infection and this failure is attributed to faulty technique.

There seems to be no relation between the duration of the brain infection and the period elapsing before the appearance of spirochaetes in the blood of mice inoculated with the infected brain material. Thus, in the case of one mouse inoculated with brain emulsion 235 days after the mouse concerned was infected, the spirochaetes appeared in the circulation on the 6th day, whilst another inoculated with brain emulsion of a mouse on the 50th day of infection did not show spirochaetes in its blood until the 10th day.

The spirochaetes were found to persist in the spleen up to the 165th day after infection, but this persistence was not constant, as only 8 out out of 19 mice showed a residual spleen infection. Only mild infections are produced by the inoculation of infected brain material, suggesting an attenuation of the virus, but by passage into other mice normal infections are obtained.

E. H.

REMLINGER (P.) & BAILLY (J.). Comportement du *Spirochaeta hispanicum*, var. *marocanum* (souche Tétouan) dans le cerveau des animaux réceptifs et réfractaires. [**The Behaviour of** *S. hispanica* **var.** *marocana* **(Tetuan Strain) in the Brains of Susceptible and Refractory Animals.**]—*C.R. Soc. Biol.* 1930. Nov. 21. Vol. 105. No. 31. pp. 433–435. [2 refs.] [Pasteur Inst. of Morocco, Tangiers.]

The authors have inoculated guineapigs intracerebrally and in addition various refractory animals such as the fowl, pigeon and tortoise, with this strain of spirochaetes. The results of subsequent examinations show that the organisms persist in the brains of all these animals, whether susceptible or not. In refractory animals, the spirochaetes persisted approximately the same length of time as in infected blood kept in glass pipettes (about 45 days).

These results differ from those obtained with rabies virus, in which the brains of susceptible animals cease to be infective shortly after being inoculated and pass through a negative period of varying duration before again becoming infective. In refractory animals, on the contrary, the virus persists in the brain for very long periods (at least 275 days in the tortoise), and there is no negative period following the inoculation.

E. H.

KRITSCHEWSKI (I. L.). Ueber den Mechanismus der Wirkung von Wismutverbindungen auf Spirochäten. [**The Mode of Action of Bismuth Compounds on Spirochaetes.**]—*Zent. f. Bakt.* I. Abt. Orig. 1930. Aug. 28. Vol. 118. No. 1/2. pp. 16–25. [7 refs.] [Microbiol. Research Inst., Education Commissariat R.S.F.S.R., Moscow.]

The author has studied the action of bismuth compounds in mice infected with *S. recurrentis* and from his results is of the opinion that the bismuth acts directly on the organisms and not indirectly as suggested by UHLENHUTH and his colleagues [see this *Bulletin*, Vol. 26, p. 672].

A comparison is made between splenectomized and ordinary mice, infected with *S. recurrentis* and subsequently treated with bismuth-yatren A. Thirty-nine per cent. of the splenectomized mice lived in spite of the absence of this important part of the reticulo-endothelial system, as compared with 87·9 per cent. of non-splenectomized mice similarly treated. Although more of the latter survived it is pointed out that 90 per cent. of splenectomized mice succumb to the infection, whilst the mortality in non-splenectomized animals is 48·3 per cent. This difference would account for the smaller percentage of splenectomized mice that were cured by the bismuth-yatren A.

It is evident in these experiments that the absence of the spleen has not appreciably affected the therapeutic action of the bismuth compounds, and it is necessary to assume that the bismuth has a direct action on the spirochaetes independent of the reticulo-endothelial system.

E. H.

MURATET (L.) & LE GAC (P.). Contribution à l'étude de la formule leucocytaire du typhus récurrent. [**The Leucocyte Formula in Relapsing Fever.**]—*Bull. Soc. Path. Exot.* 1930. July 9. Vol. 23. No. 7. pp. 697–702. [16 refs.]

The authors give a useful summary of previous observations on this subject and finally give the leucocyte counts of 14 patients suffering from relapsing fever at Wadai, Central Africa. The number of polymorphonuclears and mononuclears did not alter appreciably, but a more detailed examination showed that the number of lymphocytes diminished whilst the mononuclears increased, and the number of polymorphonuclears increased at the expense of the eosinophiles. In addition the Arneth index was always deviated towards the left.

E. H.

REMLINGER (P.) & BAILLY (J.). Absence d'immunité héréditaire dans la fièvre récurrente marocaine. [**Absence of Hereditary Immunity in Moroccan Relapsing Fever.**]—*C.R. Soc. Biol.* 1930. June 6. Vol. 104. No. 19. pp. 460–462. [Pasteur Inst., Tangiers.]

It is well known that in laboratory animals the immunity conferred by an attack of relapsing fever is of comparatively short duration, rarely more than three or four months; consequently it is unlikely that the offspring of immune parents would show any inherited resistance. The offspring of 4 guineapigs, in which one or both of the parents had been immunized against infection with a Tetuan strain of Moroccan relapsing fever, were all found to be as susceptible as normal animals.

E. H.

VAN DEN BRANDEN (F.), DUMONT (P.) & NELIS (P.). Contribution à l'étude du metabolisme du glucose sanguin dans la spirillose expérimentale. [**Blood Sugar Metabolism in Experimental Spirochaetosis.**] —*Ann. Soc. Belge de Méd. Trop.* 1930. June 30. Vol. 10. No. 2. pp. 215–222. [3 refs.] [Central Lab., Administration of Hyg., Brussels.]

The authors used guineapigs infected with a strain of *Spirochaeta hispanica* received from Tunis. The amount of glucose in the blood was estimated by Folin and Wu's method. The examination of the blood in 26 guineapigs showed that the normal quantity of glucose is in the neighbourhood of 1 per 1,000 and does not vary more than 0·2 from this figure. Particulars are given in tabular form of 9 guineapigs infected with *S. hispanica*, whose blood was repeatedly analysed at various stages of the infection. The results show that the blood sugar content of the guineapigs remained quite unaffected by the presence of this spirochaete. E. H.

BODECHTEL (G.). Sind Recurrens- und Hühnerspirochäten auf Kaltblüter übertragbar? [**Are Relapsing Fever and Fowl Spirochaetes transmissible to Cold Blooded Animals ?**]—*Ztschr. f. Hyg. u. Infektionskr.* 1930. Vol. 111. No. 3. pp. 348–353. With 3 text figs. [7 refs.] [German Research Inst. for Psychiatry, Munich.]

Lizards inoculated with *S. recurrentis* showed spirochaetes in their blood 3 days later and those inoculated with fowl spirochaetes were positive to direct examination after 48 hours. Tortoises similarly inoculated never showed parasites in the circulation, but their blood was proved to contain the infection by subinoculation into other animals respectively 24 hours in the case of *S. recurrentis* and 3 days in *S. anserina*, after the tortoise had received the original inoculation. Frogs could readily be infected with both species of spirochaete, by inoculation into the dorsal lymph sac ; the spirochaetes were very numerous in the blood. The blood of goldfish inoculated with *S. recurrentis* remained infective for 5 days, as tested by inoculations into mice, and the blood of those inoculated with *S. anserina* was infective after 4 days. Also the tissues of the fish, such as the heart muscle, were found to be invaded by the spirochaetes.

As the author points out, the spirochaetes merely persisted in these various cold-blooded animals for a few days and there was no evidence of their multiplication. E. H.

LEVADITI (C.) & PO (Li Yuan). Cycle évolutif du *Treponema pallidum*, du *Spirochaeta pertenuis* et du *Spirochaeta cuniculi*. [**The Developmental Cycle of** *S. pallida*, *S. pertenuis* **and** *S. cuniculi*.]—*C.R. Soc. Biol.* 1930. June 27. Vol. 104. No. 22. pp. 736–740. With 3 text figs. [5 refs.]

The authors have examined sections of tissues containing these spirochaetes stained by Dieterle's silver impregnation method. In all three species the organisms were found to show the same stages of development, first becoming more or less tightly coiled up into a ball, and subsequently breaking up into almost ultramicroscopic granules. These stages were found not only in lesions of the skin and mucous membranes, but also in the brain in cases of general paralysis. E. H.

MAZZA (Salvador). Espiroquetosis apendiculares. [**Appendicular Spirochaetosis.**] —*Bol. Inst. Clin. Quirúrg.* Buenos Aires. 1930. Vol. 6. Nos. 44–45. pp. 328–335. With 3 text figs. [11 refs.]

The author states very clearly the evidence regarding the pathogenicity which some observers claim for spirochaetes (*S. eurygyrata*) in the appendix.

They may be seen in the faeces of persons apparently in perfect health; others with diarrhoea, colic, and signs referable to the appendix, also excrete them and when the spirochaetes disappear after treatment, the symptoms clear up. Experiment has failed to prove that they are causative. He examined 394 excised appendices and found spirochaetes in 38 (9·60 per cent.). In 26 of them no other parasite was present; in the remaining 12 were Blastocystis or Trichomonas or both. A fairly constant accompaniment was a local fibrosis with excess of eosinophiles, intrafollicular haemorrhage, and a certain degree of atrophy of the mucosa and submucosa, but as all were cases of chronic appendicitis he sees no reason for connecting these changes aetiologically with the presence of the spirochaete. In short, since they are often present in the appendix but absent from the faeces, he concludes that there is no ground for speaking of a spirochaetal appendicitis, the condition being merely an "appendicular spirochaetosis."

H. H. S.

VASSILIADIS (P.) & JADIN (J.). Influence du *Spirochète hispanicum* sur l'infection à *Trypanosome rhodesiense*. [**Influence of** *S. hispanica* **on** *T. rhodesiense* **Infection.**]—*Ann. Soc. Belge de Méd. Trop.* 1930. June 30. Vol. 10. No. 2. pp. 133–136. [8 refs.] [Bact. Lab., Univ., Louvain.]

White mice containing large numbers of *S. hispanica* in their blood were inoculated with *Trypanosoma rhodesiense*. The incubation period of the latter infection was slightly prolonged and also the death of the animal was a little delayed. Similar results were obtained in guineapigs where the incubation period was delayed three or four days at the most, and in certain cases the number of trypanosomes in the blood was reduced.

The results show that the action of *S. hispanica* on *T. rhodesiense* is very much less marked than the action of *S. duttoni* on *Trypanosoma pecaudi* in which the incubation period in mice is prolonged from 2 days up to 12, or in one instance 19 days.

E. H.

YAKIMOFF (W. L.) & RASTEGAIEFF (E. F.). Die Spirochätose der Hühner im Nordkaukasus. [**Fowl Spirochaetosis in the North Caucasus.**]—*Zent. f. Bakt.* I. Abt. Orig. 1930. June 26. Vol. 117. No. 4/5. pp. 223–240. With 1 text fig. [78 refs.]

A general account of fowl spirochaetosis based on the study of a strain of the disease from Pjatigorsk. In that region the infection is transmitted by *Argas persicus*, and *Dermanyssus avium* cannot be incriminated.

The incubation period in fowls after being bitten by infected ticks is 4 to 7 days, and after blood inoculation 2 to 4 days. The appearance of spirochaetes in the blood is accompanied by an increase in the number of polymorphonuclears, which may rise from 20 to 30 per cent. up to 70 to 80 per cent. of the leucocytes. The strain of spirochaetosis at Pjatigorsk was found to be serologically identical with a Brazilian strain and also with other European strains. Pigeons were found to be resistant against both the injection of blood containing spirochaetes and the bites of infected ticks.

Various substances were tested as regards their action on the disease. In addition to atoxyl, spirocid, osarsol and arrhenal were found to be curative; sodium-bismuth citropyroborate had a slight effect; bijochinol, a bismuth iodine and quinine preparation, gave unsatisfactory results, as also albargin and ichthargan, two preparations containing silver.

Fowls were immunized by the injection of a chemotherapeutic agent at the same time as the virus, the best results being obtained by the use of atoxyl or spirocid.

E. H.

NICOLLE (Charles) & ANDERSON (Charles). Rapport sur les spirochètes des fièvres récurrentes transmises par les tiques. [**Relapsing Fever Spirochaetes transmitted by Ticks.**]—*Bruxelles-Méd.* 1930. Aug. 17. Vol. 10. No. 42. pp. 1140–1144.

A general summary containing nothing original.

E. H.

POU (R. Pino). Notes sur quelques spirochétoses humaines observées au Venezuela. [**Notes on Some Human Spirochaetoses observed in Venezuela.**]—*Rev. Sud-Américaine de Méd. et de Chirurg.* 1930. June. Vol. 1. No. 6. pp. 585–594. With 4 text figs.

A useful summary of the information on this subject.

E. H.

MARMO (Achille). Primi casi di pseudo-tubercolosi da spirochaeta bronchialis del Castellani in Eritrea.—*Ann. di Med. Nav. e Colon.* 1930. Nov.–Dec. Year 36. Vol. 2. No. 5/6. pp. 649–672. With 3 charts in text & 17 figs. on 6 plates. [2 pages of refs.]

RAYBAUD (A.). La fièvre récurrente hispano-marocaine.—*Marseille-Méd.* 1929. Dec. 15. Vol. 66. No. 35. pp. 760–776. [5 pages of refs.]

TALICE (R. V.). Trois ans de pyrétothérapie par le *Treponema hispanicum* en Uruguay.—*Rev. Sud-Américaine de Méd. et de Chirurg.* Paris. 1930. Apr. Vol. 1. No. 4. pp. 353–360. [15 refs.]

LEPTOSPIROSIS.

UHLENHUTH (P.). Zur Epidemiologie der Weilschen Krankheit mit besonderer Berücksichtigung der Wasserinfektion. [**The Epidemiology of Weil's Disease with Special Reference to Water Infection.**]—*Muench. Med. Woch.* 1930. Nov. 28 & Dec. 5. Vol. 77. Nos. 48 & 49. pp. 2047–2049 ; 2098–2102. [Hyg. Inst., Univ., Freiburg i.Br.]

After a general survey of the literature the author is of the opinion that the numerous cases of water infection with this disease are the result of the contamination of the water by the urine of infected rats. Consequently the rat is to be regarded as the general carrier of this disease, and the various *epidemics* at bathing establishments that have been recorded are considered to be originally due to the presence of these animals. The disease does not seem to be transmitted directly from man to man by contact, or by the agency of any insect, but is acquired either by contact with infected rats or with water contaminated by them.

Among rats the infection may be spread by direct contact from rat to rat, probably through the agency of contaminated water, and also through the ordinary water spirochaete becoming adapted for life in the body of the rat. It is also possible that some of the cases of human infection from baths may be the result of changes in the water spirochaete without the agency of rats. Further information is wanted as to the conditions under which the true pathogenic *L. icterohaemorrhagiae* can exist outside its hosts, and also the conditions causing changes in the pathogenicity of the water spirochaete and whether or not rats are necessary for such changes.

E. Hindle.

BIJL (J. P.) & KORTHOF (G.). Ueber das Vorkommen der Weilschen Infektion in Holland während der Jahre 1924–1930. [**The Incidence of Weil's Disease in Holland during 1924-1930.**]—*Arch. f. Hyg. u. Bakt.* 1930.. Oct. Vol. 105. No. 1. pp. 29–34. With 1 map in text. [Central Lab., Utrecht.]

This article supplements SCHÜFFNER'S on the same subject [this *Bulletin*, Vol. 27, p. 118]. The authors have observed 62 cases during the past six years, of which 41 occurred during August, September and October. Although the infection seemed to be usually acquired by bathing in ditches and canals, in some cases it must have been acquired from some source other than contaminated water. About 20 per cent. of the wild rats at Utrecht and Gouda were found to be infected and it seems possible that these animals are responsible for the contamination of water in the canals. In Holland, especially in the regions where canals and ditches are most abundant, there are at least 20 cases of Weil's disease every year, which can be diagnosed by the presence in the blood of the patients of agglutinins against *L. icterohaemorrhagiae.* Epidemics of jaundice may occur which are not due to this organism, but they are easily distinguished by this test.

E. H.

FISCHER (I. A.). Over het voorkomen van de ziekte van Weil op Banka. [**Weil's Disease in Banka.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1930. Sept. 1. Vol. 70. No. 9. pp. 915–923. [12 refs.]

Four cases of Weil's disease on Banka island, two of which (in Chinese) are described. In other suspected cases the leptospira could not be found. The author quotes what is known about virus reservoirs in the Dutch East Indies. The theory of waterborne infection generally accepted in Europe is not quite established in the East. The author's own research on leptospira-carrying animals in Banka showed leptospira in 6 out of 89 specimens of *Mus rattus* (6·5 per cent.) and in 22 out of 111 of *M. decumanus* (20 per cent.). The waterside habits of the latter species probably account for the higher degree of infestation. The author found leptospira in streams polluted by drains from the market and in pools near the tin mines. Anyhow, water infection is certainly possible. Other possibilities yet exist, e.g., infection by contact with the dog, known to be a virus carrier and living in a very familiar way with the Chinese population of the island.

W. J. Bais.

TROISIER (Jean) & BOQUIEN (Yves). Spirochétose méningée d'origine hydrique. Contamination digitale. [**Meningeal Spirochaetosis of Water Origin. Digital Contamination.**]—*Bull. et Mém. Soc. Méd. Hôpit. de Paris.* 1930. July 21. Year 46. 3rd Ser. No. 25. pp. 1298–1305. With 2 text figs. [2 refs.]

A detailed account of an interesting case of infection with *L. icterohaemorrhagiae*, in which the patient showed no signs of jaundice, but severe meningeal symptoms accompanied by herpes labialis, and marked injection of the conjunctivae. The inoculation of blood and cerebrospinal fluid into guineapigs gave negative results, but the inoculation of urine resulted in a typical and fatal attack of spirochaetal jaundice. Moreover, the patient's serum agglutinated *L. icterohaemorrhagiae* in dilutions of 1 : 30,000.

The patient was a man who washed dishes at a restaurant and was continually plunging his hands into filthy water. A fortnight before his attack he had wounded his hand, which might have facilitated the invasion of the spirochaetes. In support of this hypothesis the authors mention that there was a marked enlargement of the epitrochlear gland, which had to be removed on the eleventh day of the disease. The histological examination of this gland showed the presence of phagocytosed neutrophile polynuclears, which has been found in other cases of spirochaetosis without jaundice.

E. H.

KORTHOF (G.). Infectie met *Leptospira icterohaemorrhagiae* bij den hond. [**Infection of Dogs with** *L. icterohaemorrhagiae.*]—*Nederl. Tijdschr. v. Geneesk.* 1930. Aug. 16. 74th Year. 2nd Half. No. 33. pp. 4097–4103.

It was in 1916, only a year after the discovery of the *Leptospira icterohaemorrhagiae*, that UHLENHUTH and FROMME described a case of infective jaundice in a dog with leptospiras in the liver and suggested the possibility of a connexion between the disease in the dog and Weil's disease in man. OKELL, DALLING and PUGH (1925) produced positive proof by guineapig test and preparations of the kidney of the existence of infective jaundice among dogs in England. Quite recently (this *Bulletin*, Vol. 27, p. 707) KOUWENAAR and WOLFF found 6 per cent. of normal dogs in Medan, Sumatra, to be carriers of leptospiras, which,

however, they considered were not absolutely identical with those of Weil's disease. The present author has made an examination of 131 dogs by test of their sera against Weil leptospiras and by attempted culture of leptospiras from the kidney tissues. Agglutination and lysis reactions of 1 in 100 and higher were obtained in 44 out of 122 dogs but cultivation of spirochaetes was not successful. The older the dog the more certain was it that its serum would react to Weil leptospiras.

Material from dogs suspected of suffering from leptospirosis was investigated. With their sera agglutination and lysis titres to Weil leptospiras were obtained as high as 1 in 25,000 and 1 in 50,000. Guineapig tests with blood, urine, liver, or kidney were made in 9 cases with 3 positive results. These and other experiments served to raise the question whether and for how long the dog might be infective for human beings. As there is good evidence that the urine does not contain spirochaetes for much more than a week after clinical recovery, the danger of infection seems to be slight. The author comes to the conclusions that most dogs suffer sooner or later from leptospirosis, as is evident from tests of their serum, and that these infections, unlike those in rats, are of little importance for human beings because it is seldom that leptospiras are excreted in the urine.

W. F. Harvey.

FORSYTH (W. Leonard) & GOHAR (M. A.). **Leptospira in the Rat in Egypt.**—*Jl. Trop. Med. & Hyg.* 1930. July 1. Vol. 33. No. 13. p. 191. [6 refs.]

The authors examined the kidneys of 64 rats caught in the neighbourhood of Kasr el Ainy Hospital, Cairo, and found leptospira, in small numbers only, in two individuals. Guineapigs inoculated from these remained uninfected one month afterwards. Hitherto there has been no record of Weil's disease, confirmed by laboratory investigation, in Egypt, and apparently the fellaheen live in close association with the rat without danger of leptospiral infection.

E. H.

ZIMMERMANN (E.). Verbreitung und Methodik des Nachweises der *Spirochaeta icterogenes* unter wilden Ratten. [**The Distribution and Method of testing** *S. icterohaemorrhagiae* **in Wild Rats.**]—*Zent. f. Bakt.* I. Abt. Orig. 1930. Dec. 8. Vol, 119. No. 1/2. pp. 74–77. [13 refs.] [Hyg. Inst., Univ., Freiburg i.Br.]

The serum of 85 wild rats caught in Freiburg was tested for the presence of agglutinins and lysins against a " Berlin Hlld." strain of Weil's disease ; 37 gave a positive reaction and 48 were negative. The kidneys of 32 serologically positive rats were inoculated into guineapigs ; 24 became infected whilst 8 remained free from spirochaetes. The kidneys of 13 of the inoculated rats were tested both by inoculation into guineapigs and dark ground examination. There were 11 positive and 2 negative results from the guineapig inoculations, but 8 positive and 5 negative by microscopical examination. Fifteen serologically negative rats were also inoculated and of these two became infective to guineapigs whilst the others remained uninfected. The results indicate the high percentage of rats in Freiburg that are infected with *L. icterohaemorrhagiae*.

E. H.

HIGUCHI (Shusuke). Ueber die Verteilung der *Spirochaeta* (*Leptospira*) *icterohaemorrhagiae* im Rattenkörper und deren Ausscheidungsweg. [**The Distribution of** *L. icterohaemorrhagiae* **in the Rat and the Manner in which they leave its Body.**]—*Trans. Japan. Path. Soc.* 1930. Vol. 20. pp. 550–553. [9 refs.] [Med. Faculty, Imperial Kyushu Univ., Fukuoka.]

Rats were infected with *L. icterohaemorrhagiae* either by feeding them with the liver and kidneys of an infected guineapig, or by percutaneous or intraperitoneal inoculation. After intervals of two to ten days, rats were killed and their organs examined for spirochaetes. It was found that on the fourth and fifth day of the disease spirochaetes were present in the liver, kidney, spleen, adrenals, lungs, pancreas, heart muscle, ordinary muscle, the wall of the small intestine, and the lymph glands. They were always absent from the saliva and salivary glands. They soon disappeared from all organs except the kidney, although they persisted longer (up to 7 or 8 days) in the liver and heart muscle than in other organs. The blood was infected in 79 per cent. of the rats, and in some individuals spirochaetes were found on the 24th day of the disease. The urine first showed spirochaetes on the 4th day and, like the kidney, remained infected in each individual as long as it was under observation. E. H.

TROISIER (Jean) & BOQUIEN (Yves). Spirochétose ictéro-hémorragique spontanée du cobaye. Contamination de cage par un porteur de germes. [**Spontaneous Spirochaetal Jaundice of the Guineapig. Contamination of the Cage by a "Carrier."**]—*C.R. Soc. Biol.* 1930. July 11. Vol. 104. No. 24. pp. 930–932. [3 refs.]

A guineapig in the authors' laboratory succumbed to typical spirochaetal jaundice without ever having been inoculated. The infection was traced to another guineapig living in the same cage which had been twice inoculated with *L. icterohaemorrhagiae* but had shown no signs of infection. However, apparently it had developed a latent infection and was passing out virulent spirochaetes in its urine eight weeks after the first inoculation and seven weeks after the second. These organisms seem to have been responsible for the infection observed and it is thought probable that they multiplied in the damp straw and that infection was produced from this contaminated material. E. H.

DE LAVERGNE (V.), ROBERT-LÉVY & KAISER (M.). Durée d'évolution de la spirochétose ictéro-hémorragique chez le cobaye. [**Duration of Weil's Disease in the Guineapig.**]—*C.R. Soc. Biol.* 1931. Jan. 8. Vol. 105. No. 36. pp. 939–940.

Guineapigs were infected with *L. icterohaemorrhagiae* in various ways to test the influence of the method of inoculation and the dose employed. The duration of the infection was the same (8 to 9 days) in animals inoculated either intraperitoneally, subcutaneously, or by scarification, but in guineapigs infected by the ingestion of infected material the disease showed an average duration of 12 days. When the dose of spirochaetes was increased, the average duration of the infection was shortened. E. H.

BESSEMANS (A.) & THIRY (U.). Contribution à l'étude des leptospires aquicoles. [**Contribution to the Study of Water Leptospira.**]—*Rev. Belge Sci. Méd.* Paris. 1930. Dec. Vol. 2. No. 10. pp. 841–856. [1 ref.] [Inst. of Hyg. & Bact., Univ., Ghent.]

A useful summary of the authors' experiments and views on this subject, based in the main on their previous publications [see this

Bulletin, Vol. 26, p. 671, and Vol. 27, pp. 125 & 706]. The comparative characters of *L. icterohaemorrhagiae* (Pettit's strain) and the authors' spontaneous mouse leptospira (SS) and of 5 strains isolated directly from various waters in Eastern Flanders are shown in the following table :—

Resemblances.	Differences.	
	Mouse leptospira (SS) and water strains.	*L. ictero-haemorrhagiae.*
Morphology in culture.	No growth in Vervoort No. 2 containing 10 per cent. bovine or human serum.	Grows freely in Vervoort No. 2 containing 10 per cent. bovine or human serum.
Staining characters.		
Development in Vervoort's medium without serum.	Abundant growth in Walker's medium.	Grows only with difficulty in Walker.
Abundant growth in Vervoort's No. 2 containing 10 per cent. rabbit serum, ascitic fluid or hydrocele fluid.	Cultures in Vervoort and rabbit serum forming a band.	No band formed in cultures in Vervoort and rabbit serum.
Filterability.	Infection in mice producing various symptoms resembling Weil's disease, and even death.	Infection in mice producing no morbid clinical manifestations.
Formation of agglutinated masses under the influence of rabbit serum.		
Subcutaneous inoculation of mice can render their urine indefinitely infected with spirochaetes which are short and slender.	Guineapig not susceptible.	Guineapig very susceptible.
Successful treatment with tartrobismuthate of K or Na.		
Non-specific agglutination.	Specific lysis by immune serum.	Specific lysis by immune serum.

E. H.

DE ARAUJO (Eduardo). Sobre amostras de leptospira insuladas de agua na Bahia. [**Strains of Leptospira isolated from Bahia Water.**]—*Brasil Medico.* 1930. Dec. 13. Vol. 44. No. 50. pp. 1386–1389. English summary.

The author has cultivated two strains of *Spirochaeta biflexa* from water samples at Bahia. One of these strains grew near the top of the culture medium, whilst the other was localized near the bottom. The spirochaetes of the latter were more active and smaller than the former. Neither of the strains was pathogenic for guineapigs.

E. H.

INOUE (S.). Beiträge zur Rieckenbergschen Reaktion. [**Studies on the Rieckenberg Reaction.**]—*Zent. f. Bakt.* I. Abt. Orig. 1930. June 3. Vol. 117. No. 1/3. pp. 80–86. [18 refs.] [Bact. Inst., Med. Faculty, Kanazawa.]

The author has studied this reaction in various strains of spirochaetes including *S. duttoni* and various leptospira. The reaction was found to take place not only at 37° C. but also at room temperature, and to reach its maximum after 20 to 30 minutes. It was found to last from 2 to 3 hours up to as long as 7 to 15 hours. It is necessary for the spirochaetes to be healthy and active as the reaction is not shown by dead organisms ; blood platelets, bacteria or various fine particles may be used as indicators.

The antibodies on which this phenomenon depend are present not only in the plasma but also in the serum ; they are completely destroyed by heating at 72° C. for 30 minutes. With *S. duttoni* in mice the titre of these immune bodies was 1 : 10–20, and in rabbits 1 : 60–90. An immunized mouse was found to contain antibodies for 3 to 4 months, whilst a mouse that had been injected with a dose of immune serum only showed them for 2 days. In the ice chest, serum was found to preserve its properties for more than 3 months. These antibodies may be produced by the injection of dead spirochaetes. They are capable of passing through the placenta, so that the blood of the offspring of immune animals also contains the antibodies. They were also found in the milk, but not in the urine. Six strains of leptospira were examined by serological methods, including this reaction, and found to fall into three groups: *L. icterohaemorrhagiae* including L. icteroides ; the spirochaete of Akiyami type A ; and *L. hebdomadis*, including the spirochaete of Akiyami type B.

The sera of 5 patients infected with Weil's disease gave this reaction with *L. icterohaemorrhagiae* about 7 days after the beginning of the disease and after 5 months the blood still contained these antibodies. Finally the author applied the method to the examination of wild rats and gives results showing its value in the diagnosis of infection.

E. H.

ZIMMERMANN (E.). Zur serologischen Differenzierung der Spirochäten vom Weiltyp. [**The Serological Distinction of Spirochaetes of the Weil Type.**]—*Ztschr. f. Immunitätsf. u. Experim. Therap.* 1930. Vol. 68. No. 3/4. pp. 364–374. [23 refs.] [Hyg. Inst., Univ., Freiburg i.Br.]

The author has examined various strains of leptospira, using agglutination and lysis as a means of distinction. When rabbits were used for the preparation of antisera, the resulting sera reached a high titre and were extremely specific. Guineapigs produced very much weaker and less specific antisera which gave positive reactions with a considerable number of strains. When fowls were used the resulting antisera resembled those of rabbits in their strength and specificity. By means of rabbit antisera the strains studied could be divided into two groups, the first including six strains from cases of Weil's disease, and two from rats, whilst the second included four strains, Berlin R.G.A., Krommenie, Rat strain K and 221. The first two of these strains originated from cases of Weil's disease and no explanation is given of their alteration from the original type. The Rat strain K

was isolated by SCHÜFFNER from rats in Holland, and No. 221 is the Erlangen water strain after passage through guineapigs.

In addition to the strains forming these two groups, 14 water strains were examined and found to show no serological resemblance to any of the above mentioned strains. *L. hebdomadis* was also examined and found to be distinct from all the pathogenic and water strains.

It is interesting that these experiments show that there are two distinct varieties of the " Berlin " strain, the original one maintained since 1915 at the Reichsgesundheitsamt (" Berlin R.G.A.") and this strain after having been maintained at the Tropical Institute, Amsterdam (" Berlin Hlld.").

In conclusion the author expresses the opinion that the water spirochaete *L. pseudo-icterohaemorrhagiae* cannot be identified with the pathogenic form *L. icterohaemorrhagiae.*

E. H.

YANG (K.) & THEILER (Max). **The Relationship of** *Leptospira icterohaemorrhagiae* **and the Akiyami Type A Strain of Leptospira as determined by Cross Immunization Experiments in Guinea Pigs.**—*Amer. Jl. Trop. Med.* 1930. Nov. Vol. 10. No. 6. pp. 407–418. [5 refs.] [Harvard Med. School, Boston.]

The authors have made a careful series of cross immunity tests with a strain of *L. hebdomadis* known as Akiyami Type A and a strain of *L. icterohaemorrhagiae* originally isolated from rats in Boston. The virulence was maintained by passage in young guineapigs and to avoid any possible contamination the two strains of leptospira were kept in separate rooms and two different individuals looked after them.

The results clearly indicate that guineapigs actively immunized against the Boston strain of *L. icterohaemorrhagiae* are immune against the Akiyami Type A strain ; also guineapigs actively immunized against the latter are immune against the Boston rat strain. These two strains are thus shown to be indistinguishable by cross immunity tests and would seem to be the same species. In view of these results the authors raise the question as to whether it is warrantable to create a new strain just because there is some difference in the potency of the immune serum, as shown, for example, by the agglutination test. The whole subject of leptospira is becoming exceedingly involved and complex, largely due to this establishment of a number of different " species " on insufficient grounds.

E. H.

DINGER (J. E.) & VERSCHAFFELT (Françoise). Recherches expérimentales sur quelques souches de leptospires. [**An Experimental Investigation of Various Strains of Leptospira.**]—*Ann. Inst. Pasteur.* 1930. Sept. Vol. 45. No. 3. pp. 396–414. With 2 text figs. [10 refs.]

The authors obtained from BESSEMANS the Ghent water strain of leptospira which he and THIRY (this *Bulletin*, Vol. 26, p. 671 ; Vol. 27, pp. 125 and 706) had found to be pathogenic in mice. This strain was inoculated into mice at Amsterdam, but the spirochaetes never produced any infection and disappeared from the body cavity within a few hours. Moreover, they never appeared in the urine. Serologically it was found to be related to various other water strains. The authors then obtained from Dr. BESSEMANS four infected mice and these showed leptospira in the urine. Attempts to infect Amsterdam mice by the

inoculation of this urine or an emulsion of an infected kidney gave negative results. When an infected kidney was cultured in Vervoort's medium and then inoculated the Amsterdam mice became infected and showed leptospira in the urine and in rare instances also in the blood. This strain of leptospira termed "Muis Gent" was transmitted from mouse to mouse by the inoculation of blood or peritoneal exudate or cultures of the kidney. By repeated passages in guineapigs this strain gradually became virulent to these animals, but its virulence was not altered towards mice. The serological characters of the strain remained constant; it was quite distinct from various strains of *L. icterohaemorrhagiae* and also from the Ghent water strain.

Mice were then infected with a strain of *L. icterohaemorrhagiae*, and several passages made in these animals, but the serological characters remained unchanged.

Finally, the authors studied a "Slime Fever" strain that had been isolated from a human case of this disease by TERSKICH, during an epidemic in Moscow. This strain in its general growth in culture was found to resemble the water forms, but was pathogenic to guineapigs. Yet passage through these animals did not augment its virulence, and the liver contained so few organisms that they were never detected by microscopical examination. They persisted in the body cavity of mice for two days but never appeared in the urine. Serologically this slime fever strain was found to be quite distinct from all other pathogenic and saprophytic strains. It is less pathogenic than that of Weil's disease, and according to its reactions should be regarded as a distinct infection.

[This interesting paper contains the first record of a human strain of leptospira in Europe differing from the typical Weil strain both in its serological characters and in its symptoms that it produces.]

E. H.

DE ARAUJO (Eduardo). Verificações experimentaes com amostras de leptospira saprophytas. **Experiments with Various Strains of Saprophytic Leptospira.** [*Brasil Medico.* 1930. Dec. 20. Vol. 44. No. 51 pp. 1407–1411. English summary.

The author has made experiments to see if the leptospira recorded from yellow fever patients by NOGUCHI and others is an associated micro-organism, secondary invader, or *virus de sortie*. Young guineapigs were inoculated with yellow fever virus and either simultaneously or later also inoculated with various cultures of water leptospira. The blood of these guineapigs was cultured at various intervals, but in no case were leptospira recovered; consequently the author concludes that yellow fever virus has no effect on the pathogenicity of these spirochaetes. It seems probable that before the susceptibility of monkeys was discovered cases of Weil's disease were confused with yellow fever.

E. H.

RIDLON (J. R.). **Studies on** *Leptospira icterohemorrhagiae.* *Public Health Rep.* 1931. Jan. 2. Vol. 46. No. 1. pp. 1–5. [13 refs.]

The author examined 50 wild rats (*norvegicus*) caught in the neighbourhood of San Francisco, and by dark ground examination of the kidneys found 17 of them infected with *L. icterohaemorrhagiae*, this diagnosis being confirmed by the infection of guineapigs, which died with typical symptoms.

E. H.

SEMZOVA (O. M.). Zur Frage der Epidemiologie des infektiösen Ikterus. II. Untersuchung der Gewässer Moskaus auf *Spir. icterogenes.* [**Epidemiology of Infectious Jaundice. II. Examination of Moscow Water for** *L. icterohaemorrhagiae.*]—*Zent. f. Bakt.* I. Abt. Orig. 1930. Aug. 28. Vol. 118. No. 1/2. pp. 12–15. [6 refs.] [Microbiol. Research Inst., Education Commissariat R.S.F.S.R., Moscow.]

The author found water leptospira and also spirochaetes of the type of *S. recurrentis*, *S. pallida* and *S. plicatilis* in the water of the river Moskwa. No water leptospira was found in the Jausa, a tributary of the Moskwa, nor in any of the ponds of Moscow.

E. H.

RAT-BITE FEVER.

M'CLUSKIE (J. A. W.). *Spirillum minus* **derived from a Spontaneous Infection in the Mouse—with Special Reference to Pathogenicity as a Means of Differentiating it from Strains in the Rat.**—*Jl. Path. & Bact.* 1930. Oct. Vol. 33. No. 4. pp. 863–869. With 5 figs. on 1 plate. [18 refs.] [Path. Dept., Univ., & Western Infirmary, Glasgow.]

The author in the course of work with trypanosomes found a mouse infected with *Spirillum minus.* Other mice were easily infected by the inoculation of infected blood, the spirilla usually appearing in the circulation 3 to 6 days later and reaching their maximum in 7 to 12 days. The infection may persist indefinitely; splenectomy in 3 mice had no appreciable effect.

The natural transmission of this infection is very obscure, for male and female mice were kept in jars with infected individuals of the opposite sex without ever showing any signs of the disease or developing any immunity. Moreover, the offspring of infected individuals were normally susceptible, although the mice born of infected parents seemed to remain small and died sooner than controls born at the same time. 18 guineapigs were inoculated with blood from infected mice and in addition 3 human subjects, but none of them showed any signs of infection. This strain of mouse spirillum differs, therefore, from that described by SCHOCKAERT (see this *Bulletin*, Vol. 25, p. 600) which was pathogenic for man.

E. Hindle.

ROBERTSON (Andrew). *Spirillum minus* **Carter, 1887, the Aetiological Agent of Rat-Bite Fever: a Review.**—*Ann. Trop. Med. & Parasit.* 1930. Oct. 22. Vol. 24. No. 3. pp. 367–410. With 1 chart in text & 1 plate. [11 pages of refs.]

A comprehensive survey, containing an excellent account of previous work as well as the present state of our knowledge.

E. H.

SCHOCKAERT (Jos.). Antimontherapie im Rattenbissfieber. [**Antimony Treatment in Rat-Bite Fever.**]—*Deut. Med. Woch.* 1931. Jan. 16. Vol. 57. No. 3. pp. 103–104. [16 refs.] [Bact. Inst., Univ., Löwen.]

The author shows that whilst tartar emetic, stibenyl and antimosan have no obvious effect on the spirillum of this disease, stibosan has a distinct action. It is curious that stibenyl, which contains more antimony, is less toxic, and is also similar in chemical composition to stibosan, should differ so markedly in its therapeutic value in this infection. Although the spirilla as a general rule do not disappear for some days after treatment, this is also the case when arsenical compounds are used, and the author recommends the use of stibosan for the treatment of both rat-bite fever and relapsing fever, especially when there is any suggestion of these infections being arsenic-resistant.

E. H.

PREYER (Giovanna). Su tre casi di sodoku riscontrati in Roma. [**Three Cases of Rat-Bite Fever in Rome.**]—*Ann. di Med. Nav. e Colon.* 1930. Jan.–Feb. Year 36. Vol. 1. No. 1/2. pp. 10–22. With 1 text fig. [24 refs.] [Inst. of Clin. Med., Univ., Rome.]

The three cases described were typical except in the character of the temperature. The first, after an incubation period of 8 days, suffered from repeated attacks of fever lasting for two days with apyrexial intervals of 36 hours or thereabouts; the second, after an incubation period of 20 days, had febrile attacks lasting for 2, 3, or 4 days with afebrile intervals of 10–15 days; the third, after 10 days' incubation, presented an irregular fever for two days at a time with 2, 3, or 4 days apyrexia. The diagnosis was made on the history and clinical findings. On one occasion only was the spirillum found.

H. H. S.

ANGUERA (A.). Estudio clinico y experimental de la fiebre sodoku (*Spirochete morsus muris*) en España. [**Clinical and Experimental Study of Rat-Bite Fever.**]—*Medicina Países Cálidos.* Madrid. 1930. Jan. Vol. 3. No. 1. pp. 1–30. With 2 figs. & 3 charts in text. [145 refs.] French summary (4 lines).

The author describes two cases. The first was a man of 42 years, bitten on the hand by a rat. Fifteen days later he suddenly exhibited nervousness and tremor of arms and legs. Two days afterwards he shivered and the next day was violently delirious. After three days' fever the temperature fell, to rise again 48 hours later. This cycle was repeated for a month, the temperature being 41° C. in the first, 38·5°–39·7° C. in the subsequent attacks. A lymphangitis, adenitis, and a generalized macular rash were present. Neosalvarsan, 0·15 gm., injected once intravenously resulted in cure. Examination of blood smears was negative, but blood injected into guineapigs gave rise to infection and the spirillum was found, not in the animal's blood, but in the glands and organs.

The second case was a child of 7 years bitten on the nose. The wound healed, and the first symptoms declared themselves nine weeks later—local inflammation, generalized rash and attacks of fever lasting three days with apyrexial intervals of 3–4 days. This also cleared up with neosalvarsan, but three injections of 0·15, 0·3, and 0·45 gm. at five-day intervals were needed. Similar inoculation experiments were made as in the last case to determine the diagnosis. Neither patient had any complications.

H. H. S.

ANGUERA (A.). Le premier cas de sodoku en Guipuzcoa (Espagne). [**The First Case of Sodoku in Guipuzcoa (Spain).**]—*Bull. Soc. Path. Exot.* 1930. Mar. 12. Vol. 23. No. 3. pp. 266–267.

The record of a typical case of this disease. The spirillum was found in the lymphatic glands of guineapigs inoculated with the patient's blood. Although this seems to be the first record in Spain of the causative organism being found, the disease has been known since 1885 when PENA Y MAYA described a case.

E. H.

ADCOCK (E. W.). **Two Cases of Rat-Bite Fever.** [Correspondence.]—*West African Med. Jl.* 1929. Oct. Vol. 3. No. 2. p. 48.

A record of three cases of rat-bite fever in Warri, Nigeria. Two in which spirilla were found were each successfully cured by two intravenous injections of 0·3 gm. novarsenobenzol at a week's interval.

E. H.

MOCHTAR (A.). Een geval van rattebeetziekte (Sodoku). [**A Case of Rat-Bite Fever.**]—*Geneesk. Tijdschr. v. Nederl.-Indië.* 1930. Mar. 1. Vol. 70. No. 3. pp. 252–259. With 1 chart. [11 refs.]

A typical case in a native woman of 50 bitten in the hand by a rat. A guineapig and two mice were infected with the spirillum by inoculation of the patient's blood, 1, 2 and 3 days after the height of a febrile attack. If no mice are to be obtained guineapigs may serve as experimental animals, but must be injected with larger quantities (5 cc.) of the patient's blood. Two arsphenamin injections of 0·45 gm. each cured the condition.

W. J. Bais.

MURUGIAH (C.). **A Case of Rat Bite Fever.**—*Malayan Med. Jl.* 1930. June. Vol. 5. No. 2. p. 76. With 2 plates. [4 refs.]

——. **A Case of Rat Bite Fever.**—*Ibid.* Dec. Vol. 5. No. 4. pp. 149–150. [1 ref.]

The record of two cases of this disease. In both the symptoms were typical, and in the second case, although spirilla were not found by microscopical examination, the inoculation of fluid from the site of the wound into a guineapig resulted in infection with *Spirillum minus*.

E. H.

LEPROSY.

MAYER (T. F. G.). **Report of the Nigerian Branch, British Empire Leprosy Relief Association, 1929.**—*Ann. Med. & San. Rep. Nigeria, 1929.* Appendix H. pp. 95–99.

The printing of this report twice over on consecutive but differently numbered pages is presumably a printer's error rather than an emphasis of its importance. The 25 treatment centres in Nigeria in 1928 were increased in 1929 to 51, and the lepers under regular treatment rose from 2,975 to 3,630; an increasing number of these are early cases favourable for treatment; 124,400 doses were used at these centres alone, hydnocarpus oil and alepol being given, and literature on the treatment was distributed. [A recent letter from Dr. Mayer to the reviewer states that there are now some 6,000 lepers under treatment, all on a purely voluntary basis.]

L. Rogers.

MAYER (T. F. G.). **The Distribution of Leprosy in Nigeria with Special Reference to the Aetiological Factors on which it depends.**—*West African Med. Jl.* Lagos. 1930. July & Oct. Vol. 4 Nos. 1 & 2. pp. 11–15; 23–31. With 10 maps. [24 refs.]

This is a long but interesting paper by the whole time leprosy medical officer of Nigeria, illustrated by ten maps. The information was collected by personal tours and by replies to a questionary. The highest rates occur in the southern wet zone, and the least in a central elevated area. The present distribution is considered to have been influenced by the former slave trading and by previous immigration movements. Native methods of prophylaxis also had a powerful influence, and the rarity of the disease in the Yoruba area is attributed to the primitive custom of killing leper children and driving older ones into the bush, and to the isolation of lepers in other areas. In the badly affected Mohammedan areas of the north, on the other hand, there is no fear of the disease or precaution against infection. The low rate in the elevated central area is attributed to isolation from other areas and to less debilitating malaria. Famines kill off many lepers, and the Mohammedan fast causes predisposing debility, as does deficient diet in some areas, want of cleanliness, the prevalence of yaws and syphilis, sexual promiscuity, and helminthic infections.

L. R.

PANETH (O.). Ueber Leprabekämpfung in Niederländisch-Indien. [**Leprosy Control in Dutch East Indies.**]—*Seuchenbekämpfung.* Vienna. 1930. Vol. 7. No. 2. pp. 139–146. With 5 text figs.

The author discusses the leprosy problem in the Dutch East Indies and points out that wholesale compulsory segregation of the lepers in the scattered islands is quite impracticable geographically and financially, since they must number over 25,000; the problem is a very difficult one. He goes on to describe the leper asylum at Lau Si Momo in a good climate, where the inmates live a natural life and leper patients are allowed to marry; children are few, and after being weaned they are sent to healthy relatives. The patients build native huts and cultivate crops, half of which remain their own property.

Difficulties arise in getting lightly affected patients to reside with advanced ones in the institution, but the Norway system of home isolation is not practicable in these backward people. He concludes by advising compulsory segregation where practicable under comfortable conditions which will make the patients willing to remain, and also separate establishments for ambulatory and early cases, with the provision of hygienic forms of work. L. R.

HIROSE (Y.). Statistische Beobachtungen ueber die Leprakranken während der vergangenen 20 Jahre. (Ueber die Lepra auf der Insel Kyushu. 6. Mitteilung.) [**Leprosy on the Island of Kyushu, Japan.**]—*Japan. Jl. Dermat. & Urol.* 1930. Nov. Vol. 30. No. 11. pp. 1138–1161. [10 refs.] [In Japanese. German summary. pp. 120–121.] [Dermat. Urol. Clinic, Imperial Univ., Kyushu.]

The author records that in his skin clinic during twenty years he has seen 3,487 lepers (3·9 per cent. of the total patients), of whom 54·3 per cent. were macular cases, 6·9 nodular, 15·0 nerve and 19·8 of the mixed type. Men numbered 2,465 and women 653. The macular type has shown a steady increase and the nerve type a decrease. There was an important increase of the disease from the age of puberty, especially from 21 to 25 years of age, while the females showed a small increase at 50 to 55 years of age. Peasants from the country suffered most, and many patients admitted that their fathers suffered from the same disease. L. R.

NEFF (E. A.). **Report on the Central Leper Hospital, Makogai.**—*Fiji Ann. Med. & Health Rep. for Year 1929.* pp. 48–59. With 9 figs. on 6 plates & 1 diagram.

The report opens by recording that "the optimism, previously recorded as prevailing amongst patients and staff, continued during 1929." A hurricane did much damage in December and stripped the fruit from the young *H. wightiana* trees, but they fortunately survived. The admissions numbered 88, 30 died, 5 were discharged as not lepers, and 20 on parole as recovered. Indian patients are accumulating, since they now refuse repatriation, and they are more affected than Fijians. At the end of the year the total inmates numbered 442, and 373 received regular treatment. Dr. Neff states "I strongly favour the sodium salts," including alepol, but esters were also used, often alternately with sodium gynocardate. The esters of the local Ol. Calophyllum Bigator continued to prove of value in relieving nerve pains. Good illustrations of the leper colony and of improvement under treatment are given. L. R.

WELDON (S. G.). **Makogai : the Central Hospital for Leprosy in the Western Pacific.**—*Jl. Roy. Nav. Med. Serv.* 1930. Oct. Vol. 16. No. 4. pp. 264–269. With 3 text figs. [2 refs.]

This is a brief account of a visit by a naval surgeon to the Fiji leper settlement at Makogai with very similar information to that in Dr. NEFF's report above. It includes some striking photos of patients before and after treatment. L. R.

DE VERA (Bonifacio). **Morbidity and Mortality among the Nonleper Children in the Culion Leper Colony.**—*Jl. Philippine Islands Med. Assoc.* 1930. Nov. Vol. 10. No. 11. pp. 457–469. [9 refs.]

This inquiry has shown that leprosy incidence was much higher from 1922 to 1926 than from 1927 to 1929 since the children have regularly been removed from contact with their parents at a progressively younger age. The infant mortality has also fallen greatly with improved medical care. Infantile beriberi disappeared since vitamin B was added to the diet. Since 1928 no new case of leprosy has been reported among the children born in the colony.

L. R.

COPANARIS (Ph.). La lèpre en Grèce. [**Leprosy in Greece.**]—*Bull. Office Internat. d'Hyg. Publique.* 1930. Nov. Vol. 22. No. 11. pp. 2131–2134.

Leprosy has been known in Greece since the days of Hippocrates and is now prevalent in isolated centres on the coast and on islands, especially Crete, but the exact number is not known. The formation of leprosaria in Crete and Samos is said to have reduced its prevalence. During the last four years inquiries have localized the disease as described in detail in this paper and 426 lepers are now isolated, mostly at Spinalonga, Crete, and from 50 to 100 probably remain free. Alepol is being used in treatment and the results are "très satisfaisants."

L. R.

KLÖVEKORN (G. H.). Der Aussatz im Rheinland. [**Leprosy in Rhineland.**]—*Strahlentherapie.* 1930. Jan. 9. Vol. 35. No. 1. pp. 172–181. [Skin Clinic, Univ., Bonn.]

After references to the early history of leprosy and to the drastic forms of segregation practised in the middle ages, the author enumerates some of the leper institutions in Rhineland from 1162 to 1492. The largest of these was at Melaten near Cologne. Here lepers were kept for a lifetime and were allowed to beg money towards their upkeep, and in Cologne two brotherhoods collected provisions for the lepers, and suspected cases were sent there for examination. Some of these institutions were rich, and at times tramps shammed leprosy to gain admittance. Many nurses were provided and the foundation of the Order of Lazarus is described, while the constitution and domestic arrangements were on a democratic basis. The well-known middle ages precautions to prevent lepers from coming into contact with the healthy are next described. Since 1700 the Melaten leprosarium has been converted into a prison or workhouse, as scarcely a single case of leprosy remained, and he attributes the absence of the disease to the rigid precautions of the middle ages. He therefore advocates the provision of lupus homes to deal with that deforming disease.

L. R.

DENNEY (O. E.). **The National Leper Home (United States Marine Hospital), Carville, La. Review of the more Important Activities during the Fiscal Year ended June 30, 1930.**—*Public Health Rep.* 1931. Jan. 2. Vol. 46. No. 1. pp. 5–12. With 4 figs. on 2 plates.

This report deals with the work at Carville up to June 1930, and records that 55 new patients were admitted, 22 died, 23 were paroled with leprosy arrested and 8 more were qualified for parole, but elected to stay on because of disfigurement. Twenty patients were treated experimentally with vaccinated calf serum, and all but one improved. Mercurochrome with glucose intravenously has given good results in a few cases, high-frequency fulguration was of value in the removal of discrete nodules. Hot boracic compresses are used for ulcers. Vitamins are of value.

L. R.

LEPROSY REVIEW. 1930. Oct. Vol. 1. No. 4. 32 pp.

——. 1931. Jan. Vol. 2. No. 1. 38 pp. With 6 figs. (1 map) on 4 plates. Quarterly Publication of the British Empire Leprosy Relief Association, 29 Dorset Square, London, N.W.1.

In the October number E. MUIR deals with the early diagnosis of leprosy, and records that he had found Botelho's serological test for leprosy of little value. Reliance must therefore be placed on clinical methods, which enabled 61 per cent. of 929 cases seen in Calcutta in one year to be diagnosed as leprosy in spite of negative bacteriological examinations. Again 159, or 0·94 per cent. of 16,889 coolies examined in Calcutta, showed definite signs of leprosy, 72 per cent. of which were diagnosed on clinical grounds. Such bacteriologically negative cases can be treated at small cost with good results at clinics as not being infective: almost 100 per cent. of them can be cured by thorough treatment with disappearance of all signs for a period of years. Early cases of leprosy are much easier to diagnose than tubercular cases. ROGERS contributes a brief note on the intradermal leprolin test for the detection of early and latent cases; this is based on papers already reviewed in this *Bulletin*. A trial in Nauru Island has been arranged. Other papers deal with the examination of nasal smears, the treatment of common skin diseases complicating leprosy, and on the employment of lepers while under treatment, all subjects of practical importance to the practising leprologist.

In the January issue J. L. MAXWELL points out the danger of the spread of leprosy in Manchuria as the result of infection through immigration of some three million people from infected provinces of China in the last three years, for the annual number of lepers seen at the Mukden Mission Medical College is now tenfold that of a few years ago. A leprosy survey is urged. F. H. COOKE deals with and illustrates the Ho Leper Settlement in the Gold Coast which he founded; the number of lepers in this has increased from 4 or 5 in 1926 to 436 in 1930, all of whom have come voluntarily for the sake of treatment. Farming is carried on by the inmates and it is hoped that in future the settlement will be more or less self-supporting. A. KERR reports that Siam contains sufficient *H. anthelmintica* trees, locally known as "krabao," to supply oil for the treatment of a very large proportion of the lepers of the world. Janet MURRAY reports the establishment

of a leprosy clinic in northern Tanganyika Territory to attract lepers, "most of whom never showed themselves for fear of compulsory segregation." The numbers increased in one year from 4 to over 100, so other clinics were opened which hundreds attend each week, including many early cases; these the African is able to recognize. Alepol injections and trichloracetic acid locally is the routine treatment. The author concludes: "We are gradually learning that if we want to rid East Africa of leprosy there must be no compulsory segregation." B. MOISER contributes a practical paper on growing food and vegetables for leper patients, and R. G. COCHRANE begins a review of the leprosy problem in East and Central Africa, based on information collected during his 1930 tour, with an account of the Anglo-Egyptian Sudan, where a remarkable effort is being made by the medical authorities to stamp leprosy out of the badly infected humid southern districts; here over 5,000 lepers are now being treated in colonies and settlements, and are expected to become self-supporting as regards food within a short time. S. BJARNHJEDINSSON contributes an interesting article on leprosy in Iceland, where a humane system of isolation was introduced by EHLERS in 1896, since when the number has fallen from 250 to 37. Ol. chaulmoogra orally, ethyl esters and hydnocarpates have been used since 1907 and "the improvement in the condition of the patients was enormous." I. SANTRA deals with the leprosy campaign in India, and reports that about 1,000 doctors have been trained in Calcutta and elsewhere in modern treatment, and following surveys 12,000 lepers are being treated in clinics in Bihar and Orissa and Madras alone. MUIR deals with treatment of residual leprosy.

L. R.

VEDDER (E. B.). **A Discussion of the Evidence concerning the Transmission of Leprosy.**—*Porto Rico Jl. of Public Health & Trop. Med.* 1930. Sept. Vol. 6. No. 1. pp. 106–121. [23 refs.]

This paper contains an interesting statement of the usual arguments for and against leprosy being contracted by (1) Contagion, (2) Insect transmission, and (3) Soil infection, as recently suggested by WALKER, whose bacteriological work lacks confirmation. The inevitable conclusion is arrived at that there is no scientific proof of either view, so further experimental research is required.

L. R.

i. GOUGEROT & AUBIN. Elimination du bacille de Hansen par la muqueuse nasale macroscopiquement "saine." [**Elimination of Leprosy Bacilli by the Apparently Healthy Nasal Mucosa.**]—*Bull. Soc. Française Dermat. et Syph.* 1930. Nov. No. 8. pp. 1154–1155.

ii. JEANSELME. A propos de la communication de MM. Gougerot et Aubin sur l'élimination du bacille de Hansen par la muqueuse nasale macroscopiquement "saine."—*Ibid.* 1930. Dec. No. 9. pp. 1272–1273. [1 ref.]

i. A short note emphasizing that it is necessary to scrape the nasal mucosa, and not simply to rub it, when examining for the presence of leprosy bacilli, which may thus be sometimes found even when no lesions of the nasal mucous membrane are visible.

ii. The author agrees with the above statement, and he points out that he laid stress on the point in his first paper of 1897, when he introduced this method of examination.

L. R.

CAMPOS (Nelson de Souza). A bacillemia na lepra. [**Bacillaemia in Leprosy.**]—*Brasil-Medico.* 1930. Sept. 27. Vol. 44. No. 39. pp. 1093–1097.

The author has examined, after staining by the Ziehl-Neilsen method, smears of blood made from pricking the thumb or finger of lepers (avoiding, of course, local lesions). Of 14 cases of nodular leprosy he found bacilli in 12; the other two were doubtful, by which term he implies the presence of non-acid-fast bacillary fragments. Among 42 with mixed leprosy there were 31 positive, 9 negative, 2 doubtful; of 40 maculo-anaesthetic 12 positive, 24 negative, 4 doubtful; all of 4 nervous type were negative. The high proportion of positives among the nodular and mixed calls for measures to safeguard against blood-sucking insects.

H. H. S.

LE ROUX (J. J. du Pre). **The More Important Clinical Features of Leprosy.**—*Jl. Med. Assoc. South Africa.* 1930. Dec. 13. Vol. 4. No. 23. pp. 715–720. With 6 text figs.

The common lesions of early and moderately advanced leprosy are described and illustrated by photos. Treatment is also discussed, and in view of earlier scepticism regarding its value in South Africa it is of interest to note the author's statement that:

"The results of treatment at the Pretoria Leper Institution compare favourably with those obtained in most other countries where institutional treatment is resorted to. . . . . One hundred and twenty-eight patients, constituting more than 14 per cent. of the inmates, were probationally discharged as 'arrested' cases in March, 1930. Of these 50 per cent. were entirely 'symptom-free,' i.e., without mutilations or deformities or any disabilities."

He refers to the prejudice against diagnosing the early stages of the disease as leprosy on account of the "traditional stigma" due to "the amalgamation of ignorance, tradition and journalism into an unholy pact" so that "no medical man will be prepared to condemn a member of that same public to ostracism until he is perfectly sure of his diagnosis, and probably until he has seriously precluded his patient's chance of recovery." As this very early stage affords "the optimum results from treatment, it is indeed a pity that public opinion should withhold these benefits from its victims. To cure the public of its leprophobia, and thus rob tradition of its pound of flesh, appears to me the only possible solution to this question." For this purpose he suggests "an extremely audacious subterfuge," namely to call the early stages of leprosy by some name "not even remotely resembling or suggestive of leprosy." The patients could then be treated at home without segregation, while the more advanced infective cases are isolated as at present.

L. R.

BALIÑA (Pedro L.). Sobre seis casos clínicos de lepra. [**Six Cases of Leprosy.**]—*Semana Méd.* 1930. Sept. 11. Vol. 37. No. 37 (1913). pp. 777–786. With 3 text figs. [Faculty of Med. Sciences, Buenos Aires.]

The details of six patients are presented, mainly from the clinical aspect. The first was an Italian woman, 55 years of age, with maculae of arms, legs and face. In spite of the failure to find acid-fast bacilli in the nasal mucus and the W.R. being negative, owing to the anaesthetic character of the lesions a diagnosis of combined leprosy and syphilis was made and treatment by chaulmoogrol esters and arsenic gave good results. The second patient was a woman of 28 with maculo-anaesthetic leprosy whose father had suffered from the ulcerating nodular type, but had left the place eight years previously. The third, a man of 25, suffered from a very intractable prurigo, but had also a solitary anaesthetic macula on the right buttock. The fourth is of special interest : a woman of 20 with widespread macular leprosy. After a series of 35 injections of chaulmoogra, 5–10 cc. of the ethyl esters per dose, she had a severe attack of pneumonia. The leprous patches cleared so rapidly thereafter that it is believed that the acute febrile condition played no small part. The fifth, a man of 23, showed an enormous number of telangiectases on face, ears, upper part of the thorax, both arms and legs, with anaesthesia, W.R. strongly positive and Hansen's bacilli in the nasal mucus. Mixed treatment for leprosy and syphilis brought about great improvement. The sixth was a case of trophic ulcer of the foot.

H. H. S.

TISSEUIL (J.). Lèpre tuberculoïde primitive en Nouvelle-Calédonie. [**Primary Tuberculoid Leprosy in New Caledonia.**]—*Bull. Soc. Path. Exot.* 1930. Oct. 8. Vol. 23. No. 8. pp. 772–777. [Gaston Bourret Inst., Nouméa, New Caledonia.]

This paper contains a brief description of the appearances in eight patients out of 19 of these well-known leprous lesions among 117 cases examined in New Caledonia, where this type appears to be common. The lesions contained numerous lepra bacilli.

L. R.

TISSEUIL (J.). Splénomégalies sub-aiguës de la lèpre. [**Subacute Splenomegaly in Leprosy.**]—*Bull. Soc. Path. Exot.* 1930. Nov. 12. Vol. 23. No. 9. pp. 911–913. [Gaston Bourret Inst., Nouméa.]

A brief paper in which notes are recorded of four lepers in whom some increase was noted in the size of the spleen accompanying variable, and often slight, elevations of temperature, with weakness and anaemia. The organ diminished again under general treatment with iron.

L. R.

TISSEUIL (J.). Sur un cas de lèpre ganglionnaire. [**A Case of Glandular Leprosy.**]—*Bull. Soc. Path. Exot.* 1930. July 9. Vol. 23. No. 7. pp. 692–693. [Gaston Bourret Inst., Nouméa.]

In the examination of the family of a female leper her mother, who showed no skin symptoms of leprosy, was found to have a few acid-fast bacilli in an inguinal gland as revealed by puncture.

L. R.

DENNEY (Oswald E.), HOPKINS (Ralph) & JOHANSEN (Frederick A.). **Scleroderma-like Lesions in Lepers.**—*Southern Med. Jl.* 1930. Nov. Vol. 23. No. 11. pp. 1003–1006. With 6 text figs.

This is an illustrated article describing scleroderma-like lesions occurring in leprosy only on legs and hands, of large size and irregular shape, but with no appreciable elevation or central clearing. They are from bright red to brown with varying degrees of pigmentation and are extremely hard. They are almost completely anaesthetic, of very long duration ; they were found in 41 per cent. of 302 patients examined, and may contain lepra bacilli when these are found in other skin lesions.

L. R.

ADROGUÉ (Esteban) & SENA (José A.). Lesiones oculares en un caso de lepra a forma nerviosa. [**Ocular Lesions in a Case of Nervous Leprosy.**]—*Semana Méd.* 1931. Jan. 1. Vol. 38. No. 1 (1929). pp. 16–19. With 3 text figs.

The patient, now aged 34, a native of Almería (Spain), went to America at the age of 11, and to the Argentine 4 years later. When 23 years old he began to develop numbness, neuralgic pains and itching of the feet, and maculae appeared on the legs. Improvement followed the use of chaulmoogra but this had to be stopped owing to intolerance. Seven to eight years after the first symptoms disturbances of vision began ; these were lachrymation and inability to close the lids (other facial muscles, especially of the lips, were paretic), and when examined by the authors recently, 3-4 years after the eye symptoms started, there were superficial losses of substance of the cornea and fundus changes in the right eye, a choroido-retinitis with pigmentation and visual defects capable of rectification to one-half. Vision in the left eye was half, but could be brought to normal by the use of lenses ; the left fundus showed no change.

H. H. S.

TAKINO (Masuichi). Die pathologischen Veränderungen der Hautnerven bei Lepra. [**Pathological Changes in the Skin Nerves in Leprosy.**]—*Acta Scholae Med. Univ. Imperialis in Kioto.* 1930. Vol. 13. No. 1. pp. 1–15. With 2 coloured figs. on 1 plate. [Path. Inst., Japan. Imperial Univ., Kyoto.]

The author reports a careful histological study of the changes produced in the nerves of the skin in leprosy, and also what he regards as a hitherto undescribed normal nerve termination in the human hair. He concludes that the falling out of the hair in leprosy is due to degeneration of the nerves of the hairs through the development in them of leprous globi, mostly in the external rootsheaths. The degeneration of the nerves of the skin is a primary one due to leprous infiltration of the tissues. This is largely brought about by the formation of leprous globi between the nerve fibres, along single nerve fibres and also within the medullary sheaths, and even within the axis-cylinders themselves as demonstrated by him, for he has frequently found both medullary sheaths and axis-cylinders within leprous cell infiltrations, so the nerve degenerations stand in close relationship to the formation of the large-celled leprous globi.

L. R.

DWIJKOFF (P. P.). Zur Frage der pathologischen Anatomie und der Pathogenese der Lepra. [**The Pathogeny of Leprosy.**]—*Frankfurter Ztschr. f. Pathologie.* 1930. Vol. 40. No. 2. pp. 185–209. With 2 text figs. [30 refs.] ["Elias Metschnikow" Inst. for Infectious Diseases, Moscow.]

Histological examinations have been made of the tissues of a pauper leper who died of gangrene of the foot, while suffering from mixed leprosy, and who received no treatment for his leprosy The conclusion is come to that the lymphatic system is mainly affected by the leprous process, which starts in the reticulo-endothelial system, and the superficial lymph glands are specially affected in addition to the skin. Scattered groups of lepra bacilli may also be found in the internal organs, such as the liver, as shown in an illustration.

L. R.

LARA (C. B.). **Progress of Leprosy Treatment at the Culion Leper Colony.**—*Jl. Philippine Islands Med. Assoc.* 1930. Nov. Vol. 10. No. 11. pp. 469–480. [35 refs.]

The author points out that a statement of six years ago, that with few exceptions a leper sent to Culion never returned, is not now true, and he gives data to show that the recoveries are as numerous among the advanced Culion cases as in the newer regional treatment stations, and they should be still better under the present modified system of segregation. Since 1921 about 80 per cent. of 8,520 Culion cases have been treated intensively, chiefly with iodized ethyl esters. In recent years a better diet has improved the nutrition of the patients and helped treatment, and the routine injections are now given by nurses, which saves the time of the physicians. Between 1922 and 1929 1,355 patients have been paroled or discharged as negative bacteriologically for from 6 to 24 months, or 19·6 per cent. of all the treated cases, while 480 more negative cases were awaiting their discharge, making a total of 27·1 per cent. of the whole number, or 30 per cent. if all cases found to be negative once are included. Yet from 1906 to 1921 only 47 negative cases were released under former treatments, 46 of these having been under the Mercado method. The available records of the San Lazaro Hospital, Manila, shows 19 per cent. paroled and 25·8 per cent. negative at least once out of 1,576 treated, and HASELTINE's data on Hawaii cases from 1918 to 1921 would represent 19·7 per cent. paroled, while WILSON in Korea has reported 21 per cent. paroled and 30 per cent. apparent cures among 1,109 lepers. Among 70 fairly early Culion cases in children 67·5 per cent. have become negative, and of fairly advanced ones 54 per cent. Released patients could not be followed up to ascertain the number of relapses, but in Hawaii 12·8 per cent. of 242 paroled patients have relapsed, but such cases clear up again quickly with further treatment. Considering the advanced nature of the great majority of Culion patients these results are very satisfactory.

L. R.

i. NOLASCO (J. O.). **Histopathology of Leprosy under Local Infiltration: II, the Nature of the Yellowish Globules in Treated Lesions.**—*Jl. Philippine Islands Med. Assoc.* 1930. July. Vol. 10. No. 7. pp. 273–276. [10 refs.]

ii. NOLASCO (J. O.). **Local Effects of Infiltration with Buffered and Nonbuffered Solutions of Sodium Hydnocarpate and Alepol.**—*Ibid.* pp. 277–281. [4 refs.]

iii. SAMSON (J. G.), LARA (C. B.) & CRUZ (M. C.). **Treatment of Leprous Lesions of the Nasal Mucosa.**—*Ibid.* pp. 291–299. [3 refs.]

iv. EUBANAS (Froilan). **The Progress of Chemotherapeutic Investigations in Leprosy.**—*Ibid.* pp. 300–308. [41 refs.]

This interesting series of papers deals with various aspects of treatment by the very experienced Philippine workers.

i. Dr. Nolasco reports on the nature of the yellowish globules found in leprous tissues under local infiltration treatment with chaulmoogra ethyl esters, and the conclusion is come to that in all probability they are a modified form of the injected drug, which may thus be retained in the tissues up to twenty-two days.

ii. Dr. Nolasco also reports the results of a careful series of experiments on dogs to determine the effects of injecting various strengths of buffered and non-buffered solutions of sodium hydnocarpate and alepol into the skin of the chest and abdomen, as shown by subsequent histological sections. Evidence of inflammation and congestion with a fibro-cellular exudate were found, very similar to those produced by iodized ethyl esters of *H. wightiana* oil ; they were the same with buffered as with non-buffered solutions, and did not vary with the pH concentration. The irritant properties apparently reside in the fatty acid radicle, since control injections with salt solution did not produce them. Intracutaneous injections caused thrombosis of the larger subcutaneous vessels, which it is thought would limit the value in local treatment.

iii. Drs. Samson, Lara and Cruz report on the local treatment of leprous nasal lesions by different methods, including that of Dr. F. G. ROSE by ionization with alepol or potassium iodide, and Dr. LOWE's method of irrigating with weak permanganate solution followed by snuffing up a mixture of one part each of Hydnocarpus esters, creosote and camphor and nine parts of olive oil, and compare them with their own plan. In this they use fulguration by means of an insulated unipolar electrode, with a diathermy apparatus current, inserted just over the affected part of the nasal mucous membrane (but without touching it) until it is charred, after first thoroughly anaethetizing it with 5 per cent. cocaine. They also used a 5 to 10 per cent. solution of chromic acid solution on a cotton-tipped applicator as a swab, or both methods alternately. They report on 227 consecutive cases treated and observed for a year under their methods ; 81 per cent. under chromic acid, 66·6 per cent. with fulguration, and 53·5 per cent. of more severe cases in which both methods were used were found to be negative bacteriologically at the end of the observation. Congestions did best, ulcers next best, and nodules were most resistent. Profuse bleeding occasionally followed fulguration, but was controlled by further applications or by packing the nasal cavity. The importance of such forms of local treatment depends on the frequency in the Philippines with which nasal infections have been found after the skin is free.

iv. F. Eubanas contributes a brief historical account of the progress of chemotherapeutic investigation in leprosy. He falls into a slight

error when he states that HEISER suggested to ROGERS the "probability of isolating the active principles of (chaulmoogra) oil" and making soluble salts for injection, for ROGERS has himself recorded that he had made such an attempt several years before HEISER'S 1916 visit to him in Calcutta.

L. R.

ADVIER & PEIRIER. Mode d'action des huiles de *Caloncoba* et de leurs dérivés sur les bacilles acido-résistants. [**Mode of Action of Caloncoba Oils and their Derivatives on Acid-Fast Bacilli.**]—*Bull. Soc. Path. Exot.* 1930. Oct. 8. Vol. 23. No. 8. pp. 767–772.

The authors record some experiments on the lines of the work of WALKER and SWEENEY on the action of derivatives of Caloncoba, chaulmoogra and hydnocarpus oils in inhibiting the growth of acid-fast bacilli. They used bovine tubercle bacilli ground on Heyden-Hesse medium, with which they mixed 1 in 5 to 5,000 of the drugs, and they also used olive oil and control tubes. They confirmed the earlier results to some extent, but found that Caloncoba oil (one variety of which is the Gorli seed of West Africa) had less action than chaulmoogra and hydnocarpus oil preparations. They observed that after some time the acid-fast bacilli became granular without losing their staining properties, and they look on this as supporting the view that these oils have a therapeutic effect against the acid-fast lepra bacillus.

L. R.

CALLENS (Jos.). Traitement de la lèpre par injection d'une léproline. [**Treatment of Leprosy by Injection of a Leprolin.**]—*Bull. Soc. Path. Exot.* 1930. Nov. 12. Vol. 23. No. 9. pp. 909–911.

The author reports that in the Belgian Congo he has added to the usual chaulmoogra ethyl ester treatment injections of a leproline made by triturating leprous nodules with 60 per cent. alcohol and glycerine in equal parts and centrifuging. The supernatant fluid of two centrifuges is collected and used in doses of 1 to 2 cc. weekly subcutaneously. The results are said to be good.

L. R.

PALDROCK (A.). Zur unspezifischen Behandlung der Lepra mit Yatren-Casein, Lipatren, Leprosan und Alepol. [**Non-Specific Treatment of Leprosy.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1930. May. Vol. 34. No. 5. pp. 237–243.

Yet another paper advocating CO_2 and gold treatment, and claiming that it is the only "specific" treatment of leprosy, and that none of the other methods tried in a few cases produced "specific" results.

L. R.

CHIYUTO (Sulpicio). **Problems of the Negative Lepers.**—*Monthly Bull. Philippine Health Service.* 1930. July. Vol. 10. No. 7. pp. 321–328.

As the result of the remarkable progress in the treatment of leprosy the increasing number of recovered lepers who are discharged yearly

has raised serious problems in relation to them, especially now that in the Philippines lepers are released when they have remained negative bacteriologically for six months, but are expected to attend centres for eighteen months' further treatment. In the Cebu division 50 per cent. of these were found and 36 per cent. were reporting more or less regularly for treatment. Many of the negative cases elect to remain on at Culion owing to difficulties in obtaining employment if released, and to family ties in the settlement. The majority are desirous of work, but others get accustomed to being supported in idleness at Culion, while the unfounded fear of the public of discharged lepers creates difficulties for them. "The present segregation system was no doubt justifiable two decades ago when segregated lepers were never expected to get cured or returned to normal life," but now that "lepers under scientific and proper medical care and treatment are able-bodied men capable of performing mental and manual work," the segregation law should be amended by making able-bodied lepers work while confined in a leprosarium, and recovered lepers should earn their living as agriculturists.

L. R.

GOMEZ (Luis B.). **Preliminary Survey on Negative Lepers in the First Sanitary Division, Cebu.**—*Jl. Philippine Islands Med. Assoc.* 1930. Aug. Vol. 10. No. 8. pp. 322–326.

This paper deals with the very important and difficult question of following up recovered lepers discharged on parole after successful treatment, to determine how far the results are lasting. Of 148 such cases who were traced in one division 135 remained negative and 13, or almost 10 per cent., had become positive again. A number of patients had ceased to attend the centre for further treatment, but these were mostly living at a distance involving heavy travelling expenses. The social conditions of the discharged patients are also dealt with, and help for the families of segregated lepers when required is recommended, as well as compulsory training of boys and girls in some productive occupation while at Culion to enable them to gain their livelihood after their discharge.

L. R.

TRINCAO (Carlos). Les réactions de Kahn et de Wassermann chez les lépreux. [**The Kahn and Wassermann Reactions in Leprosy.**]—*C.R. Soc. Biol.* 1930. Oct. 16. Vol. 105. No. 27. p. 160. [2 refs.]

The Kahn and the Wassermann tests were both carried out in 61 lepers, and were in agreement in 36, or 59 per cent. In 25 in which there was disagreement 14 differed in the degree of the reactions and in these in 11 the Kahn was the more sensitive. In the remaining 11, with absolute discordance, the Wassermann only was positive in 1 case and the Kahn only in the other 10, so the latter appears to be the more sensitive reaction in leprosy. In none of the patients was syphilis suspected.

L. R.

AHUJA (M. L.) & MALLICK (S. M. K.). **Serological Diagnosis of Syphilis in Lepers.**—*Indian Jl. Med. Res.* 1930. Oct. Vol. 18. No. 2. pp. 707–710. [7 refs.] [Central Research Inst., Kasauli.]

The authors report the use of Kahn's qualitative procedure for obtaining evidence of syphilis in 100 lepers. The results were in agreement with the Wassermann test in 90 per cent. of the cases. The Kahn test is simpler to perform, but in some cases rather less easy to read.

L. R.

TÔYAMA (Ikuzo), MIYOSHI (Shukuji) & KUBOKAWA (Katsumi). **A Contribution to the Serology of Leprosy.**—*Japanese Jl. Dermat. & Urol.* 1930. Sept. Vol. 30. No. 9. pp. 887–934. [In Japanese. English summary pp. 93–97.] [Dermat. Urolog. Clinic, Imperial Univ., Tokyo.]

The authors give details of (1) a floccule-forming reaction and (2) a complement-fixation reaction, both of which they regard as of value in the diagnosis of leprosy. Confirmation or otherwise will be awaited with interest. For the technique the original paper must be consulted.

L. R.

MOLINELLI (Ernesto A.) & RÉ (Pedro M.). L'amino-acidémie dans la lèpre. [**The Amino-Acidaemia in Leprosy.**]—*Rev. Sud-Américaine de Méd. et de Chirurg.* Paris. 1930. Aug. Vol. 1. No. 8. pp. 781–785. [6 refs.] [Faculty of Med. Sciences, Buenos Aires.]

The authors tabulate details of 39 lepers in whom they investigated the degrees of amino-acidaemia. They conclude that an increase may occur in all three types of leprosy; normal figures were met with in some, but in no case was there decrease except during an exacerbation of the disease. In over half the cases a parallelism was noted between the clinical evolution of the disease and modifications in the amino-acidaemia.

L. R.

WHERRY (W. B.). **Cultivation of an Acid-Fast Bacillus from Leprosy.**—*Philippine Jl. Sci.* 1930. Dec. Vol. 43. No. 4. pp. 577–580. With 2 figs. on 1 plate. [2 refs.]

This paper is best summarized in the author's own words as follows :—

" An acid-fast bacillus was grown in a semisolid medium composed of equal parts of nutrient agar, and hen's ovomucoid and yolk, prepared by boiling in 3 to 6 per cent. glycerin solution, when there was added to this 1 or 2 drops of oleic acid and 1 or 2 drops of 10 per cent. dextrose solution for each cubic centimeter of medium. Multiplication occurred best in cultures incubated at 35° to 37° C., with little oxygen present but carbon dioxide present. Microscopic examination after four to six weeks incubation showed the presence of numerous microscopic colonies. Growth was obtained from three cases. From two of the primary cultures, subcultures were obtained at a time when the primary cultures were three and one-half months and one month old, respectively. Like the lepra bacilli in smears

from cases, the cultivated bacilli do not stain by the acid-fast method if they are first treated with xylol and alcohol."

L. R.

NORTH (Edgar A.). **An Investigation in Rockhampton into the Possible Relationship between Human and Rat Leprosy.**—*Health.* Canberra. 1930. July. Vol. 8. No. 7. pp. 67–74.

This inquiry was made at Rockhampton, Australia, in relation to the premises occupied at any time by nineteen known human lepers, since in five years 80 rats had been found infected with rat leprosy in the town. Of 15 lepers, 12 gave a history of previous contact with lepers; this is in agreement with a statement of L. ROGERS. No convincing evidence was found of any connexion between human and rat leprosy.

L. R.

SCHLOSSMANN (K.). Ueber Gleichgewichtsstörungen der Eiweisskörper im Blutserum Leprösser. [**Disturbances of Equilibrium in the Serum-Proteins of Lepers.**]—*Ztschr. f. Immunitätsf. u. Experim. Therap.* 1930. Vol. 68. No. 1/2. pp. 154–169. [24 refs.] [Bact. Inst., Tartu-Dorpat, Estonia.]

The author reports on a study of the blood serum in lepers, in which the chemical and physical alterations of the serum-albumin and serum-globulin play a prominent part. The sera of 18 lepers, 20 syphilitics and 10 healthy persons were examined by azotometrical albumin-estimation, complement fixation and the Sachs-Georgi reactions. The albumin values for normal human blood were first determined with the help of the azotometer, including the total albumin, the serum-globulin and the serum-albumin respectively, and then similar observations were made on the blood of the leprous and syphilitic subjects. The globulin fractions were not found to be uniformly increased in the leprous and syphilitic sera. The syphilitic sera showing a low albumin content generally give a strong positive complement fixation reaction, but similar leprous sera usually give negative or only weakly positive reactions. Further, the Sachs-Georgi reaction does not run parallel with the globulin and the albumin content of leprous sera, and the globulins of leprous sera are more stable and give heavier sedimentation than the globulins of syphilitic sera.

L. R.

RAJEWSKI (A. S.). Zur biochemischen Charakteristik der Lepra. [**Biochemical Changes in Leprosy.**]—*Arch. f. Schiffs- u. Trop.-Hyg.* 1930. Dec. Vol. 34. No. 12. pp. 651–657. With 1 chart in text.

The first part of this paper deals with the variations in the mineral salts in nerve leprosy. The author concludes that in stationary forms the calcium balance is maintained, but in actively progressing forms calcium salts are deficient and sodium salts are increased; but these changes do not run parallel to the severity of the disease. Milk alters the loss of calcium salts into a gain. The calcium content of the serum is usually within the normal limits.

In part 2 the carbohydrate metabolism is dealt with. He finds it to be reduced in leprosy, and he thinks this is largely due to functional disturbance of the liver.

L. R.

REVIEWS AND NOTICES.

HANDBUCH DER HAUT- UND GESCHLECHTSKRANKHEITEN. 1930. Vol. 10. Part 2. pp. xviii+907. With 220 figs. **Die Lepra** [KLINGMÜLLER (Victor) pp. 1–805; & Index pp. 843–907]. **Lepra in Literatur und Kunst.** [Leprosy in Literature and Art.] [GRÖN (K.) pp. 806–842. With 48 figs. (1 coloured)]. 1930. Berlin: Verlag von Julius Springer. [Bound Rm.146; Unbound Rm.138.]

These two works concerned with leprosy are bound together in part 2 of the tenth volume of a handbook of diseases of the skin and genitals, with many contributors and several editors whose names are given on the cover, and issued by the German Dermatological Society. Dr. Klingmüller of Kiel starts his Introduction with a definition of leprosy in general terms to the effect that it is a contagious and transferable disease, confined to man. The method of transmission is not fully known. It is generally chronic with sometimes acute outbursts, and appears in two chief forms, nodular leprosy and nerve leprosy, together with mixed forms. Most cases are incurable and death is only a question of time. There has, however, been a great improvement during the present century in methods of treatment, and cures and partial recoveries are recorded. Leprosy may appear in any land, race or climate, and at any age, and among rich and poor. All these aspects are fully dealt with and discussed in the chapters which follow with a wealth of reference to many authorities—indeed, few writers on the subject of leprosy can have escaped the author's notice. Following a history of leprosy from ancient times, there is a long section giving the geographical distribution of the disease in all continents and countries. Such figures as are given in this part are generally those referring to the present century; but history shows clearly the reduction, and almost the disappearance, of leprosy in Europe following upon segregation of lepers and improved methods of sanitation and types of living among the general population. The cause of the disease is Hansen's bacillus, discovered in 1874, an acid-fast germ extremely difficult to deal with as regards culture methods. No one has succeeded in producing a pure culture with which leprosy can be reproduced in man or animals. The bacillus is fully described as to behaviour in trial culture media and as to staining reactions and microscopic appearance, and the possible transmission to certain animals is considered. Insects as possible transmitters are put under review; but there does not seem to be any proof of such transmission. The clinical part of the book is very full and the appearances which may develop in any form of leprosy are clearly shown in the illustrations, and, apart from the diagnostic features visible in the skin, etc., the effects of infection on the blood and internal organs receive attention. Differential diagnosis and prognosis are followed by a chapter on prophylaxis. The isolation of cases is the most useful method of preventing the spreading of leprosy; but the author notes that want of money often makes it difficult to carry out this system to a perfect end. The final chapter deals with direct and indirect treatment of the disease and brings hope. Much has been done and cures have been recorded since it was discovered by Sir Leonard ROGERS that the bacilli of leprosy can be destroyed by hydnocarpates and morrhuates. The author mentions salvarsan, arsenic, vaccines, endocrine gland preparations, and even diluted snake venom. A bibliography covering 60 pages ends the book. With great impartiality and intense industry, Dr. Klingmüller has produced a work of reference which will be of great use to every medical library.

ii. Of this short essay of thirty-six pages it is not necessary to say much. Literature and Art, writes Dr. Grön of Oslo, ideal factors of culture, have both to contain the Greek standards of beauty and goodness. Besides books mentioned in the text, such as "Armen Heinrich" by HARTMANN VON AUE and "Le lépreux de la cité d'Aoste" by XAVIER DE

MAISTRE, a list of books and plays on the subject of leprosy is to be found at the end of the essay. The plates and other illustrations showing examples of leprosy in art are very well reproduced. Six of these refer to Job as a leper and belong to the sixteenth century. Two of these introduce the devil in the act of inflicting the disease upon his temporary victim. Lazarus and the dog appear in a miniature and in a picture painted about 1570. We cannot refer to all the illustrations; but one of St. Martin cutting his cloak to share it with a beggar and several referring to St. Elizabeth should be mentioned. They include a fine reproduction of Murillo's picture "The Holy Elizabeth" to be seen in Madrid.

J. H. Tull Walsh.

BOREL (E.) [Médecin Commandant des Troupes Coloniales, Chef de Laboratoire à l'Institut Pasteur de Saigon]. **Les Moustiques de la Cochinchine et du Sud-Annam.** [The Mosquitoes of Cochinchina and South Annam.] Préface de E. ROUBAUD.—423 pp. With 122 figs. & 3 plates. (Monographie III. Collection de la Société de Pathologie Exotique.) 1930. Paris: Masson et Cie. [70 fr.]

Besides the descriptive catalogue of the mosquitoes of Cochinchina and the territory immediately adjoining, which forms its bulk, this volume also includes a preface (by Prof. E. ROUBAUD) containing some biographical detail of the author, an introductory section on the topography and meteorology of the country, and an epilogical section on three of the notorious diseases transmitted by mosquitoes, namely malaria, filariasis, and dengue fever. The Preface informs us that the author, M. E. Borel, was a Surgeon-Major of the Colonial Service of France, who studied Medical Entomology in Prof. Roubaud's laboratory during a large part of the year 1924 with a view to undertaking direction of the antimalaria service of the Pasteur Institute of Saigon in Cochinchina. A local mosquito-survey, which was carried on from 1924 to 1928, was one of the fundamental operations undertaken by Borel on his return to Saigon. Some of its results have already been noticed in the *Archives des Instituts Pasteur d'Indochine*; its completed results were among the documents deposited in Roubaud's laboratory by Borel when he returned to France in 1928. Borel died in October of that year, and the publication of the results of his work in the present monographic form has been ably conducted by Prof. Roubaud, who states that he has added to it "a small complement of figures with their necessary legends" and some fine photographs illustrating some of the more typical breeding-places of anophelines.

The descriptive roll of the mosquitoes includes 95 species—25 Anophelinae; all the rest segregated as Culicinae—and occupies 340 pages. The individual descriptions of species, which include (so far as has been possible) both the adults and the larvae, are careful and concise, are liberally illustrated in text-figures of significant anatomical features, and take due note of facts of pathological significance and of local distribution. The 25 Anophelines are grouped in 2 subgenera—Anopheles and Myzomyia—and the habits and environment of their larvae, and the predilections of their adults in relation to human affairs, are set forth in a convenient and independent table.

The introductory section contains a great deal of significant meteorological detail. Of the 34 pages in the concluding section, 23 are allotted to malaria; most of these are quite pertinent to the business of the medical entomologist, but those dealing with the morbid manifestations and clinical treatment of the disease seem to be superfluous.

A. Alcock.

KIRK (J. Balfour) [M.B., Ch.B., D.P.H., D.T.M. & H., Director, Medical and Health Department, Mauritius]. **Public Health Practice in the Tropics.**—pp. xiv+498. With 80 illustrations. Churchill's Empire Series. 1931. London: J. & A. Churchill, 40, Gloucester Place, Portman Square. [15s.]

The subject matter dealt with in this volume is divided by the author into two, part I under the title "Preventive Medicine" occupying 353 pages and part II under "Sanitation" occupying the remainder of the total 487 pages. The first chapter in part I is devoted to climatic and physiological considerations, the latter including dietaries and clothing for the tropics. The second chapter deals with maternity and child welfare, and in introducing these subjects thus early in his volume the author not only assigns them a prominence which is unusual but also emphasizes the fact that the rapid development of such services will have an incalculable effect for good in spreading the knowledge and practice of hygiene among tropical peoples. The principal diseases of the tropics are set out in alphabetical order, most space being given to Plague, 54 pages, and Ankylostomiasis, 28 pages; Sleeping Sickness 27 pages, Malaria 20 pages, and little space to Yellow Fever, 7 pages, and Yaws, 3 pages.

Part II on Sanitation begins with Chapter XXII on Housing and Town Planning. School hygiene, Milk and Milk supplies, Meat supplies, Water supplies in Rural areas and small towns, Public latrines and Baths are next discussed. The question of the tube or bore hole latrines for tropical village communities might well have received some attention and discussion. Chapters on the Collection and Disposal of Excreta, on Vermin and Public Health follow. An ingenious stomoxys trap invented by M. Bourgault du Coudray is noted and figured. The book ends with chapters on Disinfection and Disposal of Refuse.

It is probable that in subsequent editions the author may find it expedient to rearrange his values somewhat. For example the subjects of malaria and yaws receive in this volume rather less space than ankylostomiasis and beriberi, and more attention might be given to village problems of sanitation. After all, the village populations of the tropics are far in excess of the urban populations, and their sanitation problems are proportionately greater.

The book is frankly a compilation, and the author is anxious and willing to acknowledge the many sources to which he is indebted. But the fact that a book of this nature is largely a compilation by no means necessarily detracts from its value. In fact it is in these days difficult for any book which covers such a vast field of work to be other than largely a compilation, and the author has been successful in producing in a compact form and in a readable manner the essential information.

D. B. Blacklock.

MAJUMDAR (Akhil Ranjan) [M.B., Bengal Medical Service, etc.]. **Bed-Side Medicine. A Hand-Book of Medical Diagnosis. Symptoms, Physical Signs, and Laboratory Methods, from Tropical Standpoint.** With a Foreword by Major-General J. W. D. Megaw, C.I.E., I.M.S., Director General of Indian Medical Service. Second Edition.—pp. xi+584. With 190 figs. 1930. Calcutta: The Book Company Limited, 4–4A, College Square. [Rs. 7–8.]

Dr. Majumdar may be congratulated on the 2nd edition of his book. It will undoubtedly be of great use to those for whom it is intended—the medical practitioner and the student of medicine in India. He has certainly covered the whole ground very thoroughly and has incorporated in the volume many of those practical hints and tips which, as he rightly says, can only be learnt at the bed-side. The book is divided into sections

each of which deals with the particular system in a very efficient manner. The section devoted to fevers is particularly good and the same remark applies to that on the alimentary system, in which, as elsewhere in the book, the author has taken pains to include much recent work. A good feature is the large number of photographs and illustrations, many of them original, which have been included. There is no doubt that this book will be of considerable assistance in differential diagnosis. Current medical literature has been freely drawn upon and has been acknowledged in a full list of references. The book is handy in size and the index is adequate. One must, however, take note of the fact that there are many misprints and defects in punctuation which should be remedied in future editions.

M. L. C. Irvine.

VAN DER MADE (Wilhelmina Maria). **Constante en inconstante eigenschappen der bloedgroepen. De verdeeling der bloedgroepen in West-Java.** [Constant and Inconstant Attributes of Blood Groups. Distribution of Blood Groups in West Java.] [Thesis for Doctorate in Medicine at the Medical High School, Batavia.]—pp. 99+x. With 10 text figs. & 1 map. Weltevreden: Druk van G. Kolff & Co.

The first two out of the three chapters which make up this monograph are devoted to what may be designated respectively the specific and non-specific features of blood groups. Under the heading of specific characterization there are discussed most of the theory and known facts of the subject. Apart from the careful description of technique the most interesting sections are those relating to the importance of blood grouping for transfusion, the inheritance of blood groups, and their inclusion in the scheme of Mendelian transmission. Questions of alleged paternity may be capable of settlement by means of blood tests. The author expounds the subject of the distribution of blood groups in human progeny in great detail from a Mendelian point of view. Another side to this question of inheritance is that of the racial distribution of blood groups. In this regard it was determined by the HIRSCHFELDS that the A grouping was characteristic of, and predominant in, European peoples and that a B grouping prevailed in oriental races. Their biochemical "race index" is represented by the relative proportion of A groups to B groups in a test population. It is necessary, however, to include in each of these two single groups the group AB which is possessed, as regards its erythrocytes, of both A and B agglutinable characters and whose serum in consequence is incapable of agglutinating either A or B erythrocytes. The race index (I) is given as (A+AB)/(B+AB) and has been used by the author on a large scale to determine the affinities of the component elements of the population of West Java.

In the second chapter of the work a very full discussion is entered upon as regards the physical chemistry and the colloidal chemistry of the blood so far as it affects the subject of blood grouping. It is in the third chapter that the author sets out what is essentially her own work. This ranges over a variety of subjects from the anthropological question of the presence of a population older than the predominant population and existent in the outlying parts of Java, to the correlation, if any, exhibited by body-build, disease and mental character. The general trend of this part of her research may best be realized by setting out her main conclusions. These are :—

1. The higher biochemical race index in the outlying districts of West Java indicates that a residual population is to be found there which is older than the Soendanese.

2. Sufferers from tuberculosis, lunatics and prisoners, among Soendanese, Javanese and Sumatrans, afford no evidence of any appreciable deviation from the race index.

3. No definite correlation could be found between character, body-build, finger prints and blood groups.

A further investigation had reference to possible alteration of agglutination titres. Thus it was found that repeated bleeding, within a short interval of time, seldom produces any effect on the agglutination titre of a blood-group serum and that addition of phenol or soda to serum, unless in high concentration, is without harmful action.

W. F. Harvey.

WAR OFFICE. **Memoranda on Medical Diseases in Tropical and Sub-Tropical Areas 1930.** Fifth Edition.—300 pp. With 129 figs. & 1 double plate. 1930. London: H.M.S.O. [6s.]

The first edition of this work, as stated in a short preface by the Director General, Army Medical Services, was compiled by the late Sir Andrew BALFOUR and dealt with Diseases of the Mediterranean; the two succeeding issues were also prepared by him. The fourth and present editions were entrusted to Colonel W. P. MACARTHUR, who enlarged the scope of the work to include Medical Diseases in Tropical and Subtropical Areas. This last issue is printed on a slightly smaller page and in somewhat larger type, but by judicious revision, elision and addition the whole has been brought up to date although the total of pages is increased by 23 only.

Six years have elapsed since the previous edition was published and in the interval the advance in knowledge of diseases of warm climates has been considerable, notably in blackwater fever, yellow fever, and sprue. The two last received no mention in the fourth edition, but now find place; tropical typhus is another welcome addition. Other omissions noted in the former review (this *Bulletin*, Vol. 22, p. 341), namely epidemic dropsy, leprosy and yaws, are still conspicuous, and rabies would seem to be of sufficient importance to warrant inclusion. It must have been no easy task to decide how much to include and doubtless, though we know them not, there are good reasons for these omissions.

As the arrangement of subjects in the book is alphabetical it may be well to consider certain points calling for notice in a similar sequence.

As in previous issues the term Ancylostomiasis is used to cover hookworm infestation whether by Ancylostoma or Necator. To spell it with a "k" has advantages when the condition is not restricted to the former parasite. The insertion of diseases transmitted by Simuliidae is fully warranted by the growth of knowledge on onchocerciasis. The persistence of *V. cholerae* in the gall-bladder of the chronic carrier is stated as a fact (p. 80), though this has been the subject of research and is definitely denied by some workers in India (*Indian Journal of Medical Research*, July, 1929). The inclusion of an account of acute gastro-enteritis in children in the Diarrhoea section is a good thought. Influenza, which appeared in the last edition, is now omitted, as is also Pneumonia, and rightly so, to make room for matters of more importance. Under the diagnosis of Leptospirosis (p. 157) is the statement "Yellow fever . . . may simulate leptospirosis icterohaemorrhagica." Mention might be made, either here or in the new section on yellow fever, of the fact that not mere simulation but actual cases of leptospirosis may occur in a yellow fever epidemic and, if rapidly fatal, may be mistaken for yellow fever. It is quite probable that NOGUCHI had the ill-fortune to fall in with one of these when he found what he called *L. icteroides*; if so, it indeed led him to a series of very erroneous conclusions, though he may not, initially, have been so "grievously deceived." Under Malaria to indicate those species of Anopheles which are of special epidemiological importance is of vastly greater value than the mere list given in the previous issue. A new illustration of a patient with Oriental sore has replaced one which has done yeoman service for many years in this and other text-books; though the sore is perhaps not

quite such a fine specimen, this is more than compensated for by the better plate. When speaking of the treatment of plague the author sounds a timely warning on the dangers of a " run of favourable cases " leading to the vaunting of some insufficiently tested treatment; the history of blackwater fever affords another example of the same danger. A picture of the cercaria of *Sch. mansoni* usefully replaces the three indefinite forms with poor detail depicted previously. On the subject of Veldt sore and its treatment, the use of diphtheria antitoxin will not be successful unless the true Klebs-Loeffler organism is present; for cases associated with *diphtheroid* organisms (whatever this vague term may include) it is ineffectual. The fact that *Corynebacterium diphtheriae* is associated aetiologically with the condition has been handed down from year to year and from book to book largely on clinical grounds; it is a matter for wonder that the subject is not studied intensively and set at rest once for all on definite scientific bacterial lines.

The alphabetical arrangement of the contents leads to the separation of typhoid from the paratyphoid fevers. Would it not be better to class them all under Enterica, which would then find appropriate place before Fevers in the East, which includes an excellent description of the shorter tropical fevers, full of interest and instruction? A very comprehensive general account of Sprue finds a place for the first time in this edition, together with adequate detail of the treatment most successful in the author's hands.

There is a final chapter, on Zoological Nomenclature, which will prove most useful in explaining to the uninitiated the apparent vagaries of systematists in altering names.

Misprints are exceedingly few; in the title of Fig. 46 is one; the only others likely to lead to disappointment are the numbering of figures after 119. There is here a skip of 30, so that 120–129 are numbered 150–159; the prospective purchaser may thus think that there are 28 more figures in the new edition, whereas there are two less than in the fourth.

The copy sent for review has been bound up somewhat erratically, with the result that in several instances figures fail to correspond with the letterpress and undulant fever has become mixed up with typhoid and trench fever, but these small hazards, if we may so call them, do but add zest to the reading, and the work as a whole is an achievement on which Colonel MacArthur is to be congratulated. Whereas in attempting to include so much in so small a compass there might have resulted a mere dry catalogue of aetiology, symptoms, diagnosis and treatment, we have instead descriptions which are accurate, concise, and presented in a manner which make the reading a real pleasure.

H. H. S.

Anglo-Persian Oil Company, Limited. **Medical Report for the Nine Months ending 31st December, 1928** [Young (M. Y.), Chief Medical Officer].—105 pp. With 5 plates, 7 diagrams, 2 folding charts, 6 graphs & 1 folding map.

——. **Ditto for Year ending 31st December, 1929** [Young (M. Y.), Chief Medical Officer].—132 pp. With 9 plates, 18 diagrams, 2 charts & 1 folding map. [No date or place of publication stated.]

It is unusual for the larger commercial firms to publish comprehensive medical reports and much valuable information is consequently allowed to go to waste. The United Fruit Co. in the West and the Anglo-Persian Oil Co. in the East are notable exceptions. The reports reviewed here cover 9 months of 1928 and the whole of 1929, and each deals in turn with Organization and Vital Statistics, and the Medical, Surgical, Pathological, and Public Health Sections. Naturally, they deal largely with routine work, which is very heavy, but research is not neglected. Bacillary dysentery was

investigated in 1928 when a peculiar organism was isolated from a limited number of cases and the work was continued in 1929. The chief subject of research was carried out by Dr. MARSH, in 1928, on the Physiology of Heat Regulation and, in 1929, on the Etiology of Heat Hyperpyrexia. The two combined formed the subject of a paper read before the Royal Society of Tropical Medicine and Hygiene towards the end of 1930, which was followed by an interesting discussion. This paper is being reviewed separately.

Knowing that housing conditions directly influence industrial efficiency, every effort has been made by the company to provide proper housing accommodation for the staff, as an essential part of preventive medicine. In the 1929 report plans are reproduced showing types for Senior and Junior Married Bungalows, Senior Bachelor's Bungalow, and Bachelors' Flats; also for Single and Married Clerks' Quarters, and Single and Married Artizans' Quarters, the aim in each case being to ensure maximum protection from heat and spacious, well-ventilated rooms with economy of construction, but in no case have the former been sacrificed to the latter.

H. H. S.

NIESCHULZ (Otto). Surraübertragungsversuche auf Java und Sumatra. **[Surra-Transmission Experiments in Java and Sumatra.]**—*Veeartsenijkundige Med. No.* 75. *Departement van Landbouw, Nijverheid en Handel Ned.-Indië.* 296 pp. With 8 plates. 1930. Utrecht: Kemink en Zoon N.V.—Over den Dom.

In this volume the author brings together the accounts of his experiments, carried out in Java and Sumatra, on the transmission of surra. In his introduction he points out that the discovery of Naganol has made it possible to reduce the incidence of the disease in the equine population because the drug can be used as a prophylactic. This does not, however, assist in its eradication because the reservoir of the infection is in apparently quite healthy buffaloes.

For the proper control of surra a first essential is an accurate knowledge of the method or methods of transmission.

The present publication contains not only the experiments which have already been reported in Dutch journals, but a considerable number which have not previously been recorded.

The author reviews the literature and divides his review into sections dealing with the various kinds of insects used. Some thirty pages are occupied by a description of the materials and the technique employed in the experiments. Forty-three different kinds of insects have been tested, including Tabanus, Chrysops, Haematopota, Stomoxys, Musca, Anopheles, Armigeres, Stegomyia, Culex, and Cimex, and the bulk of the book is made up of accounts of the experiments carried out with these. No attempt was made to check the positive results reported by CROSS in the transmission of Surra by Ornithodorus. Ornithodorus is not known to occur in the Dutch Indies.

The author is of the opinion that Tabanidae are by far the most important transmitting agents of surra, and that the transmission is a mechanical one.

A. Leslie Sheather.